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Podrid's Real-World ECGs

A Master's Approach to the Art and Practice of Clinical ECG Interpretation

Volume 4 Arrhythmias—Part B: Practice Cases

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These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy, daughter Sapna, and son Sanjay, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack

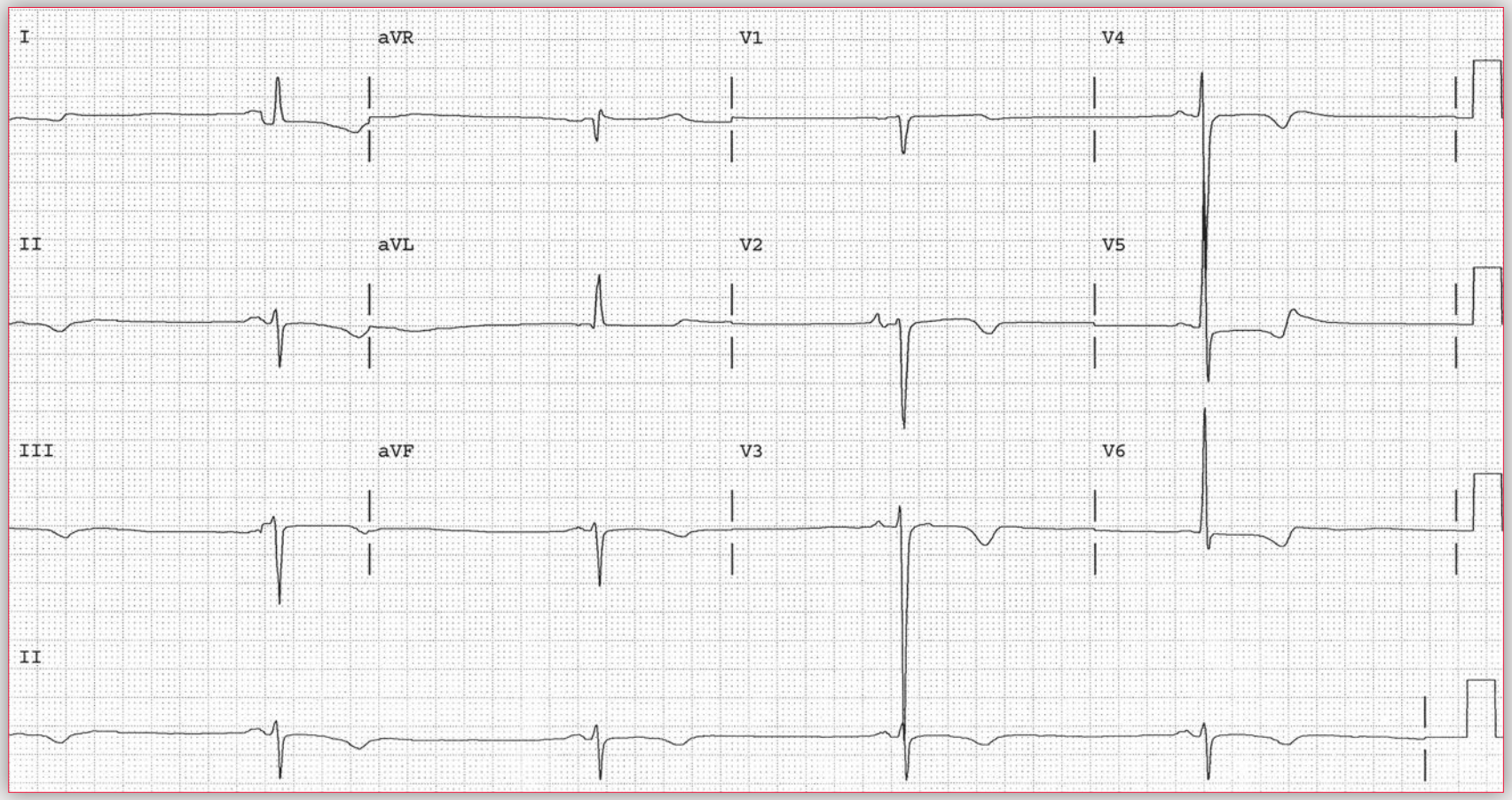
Peter A. Noseworthy

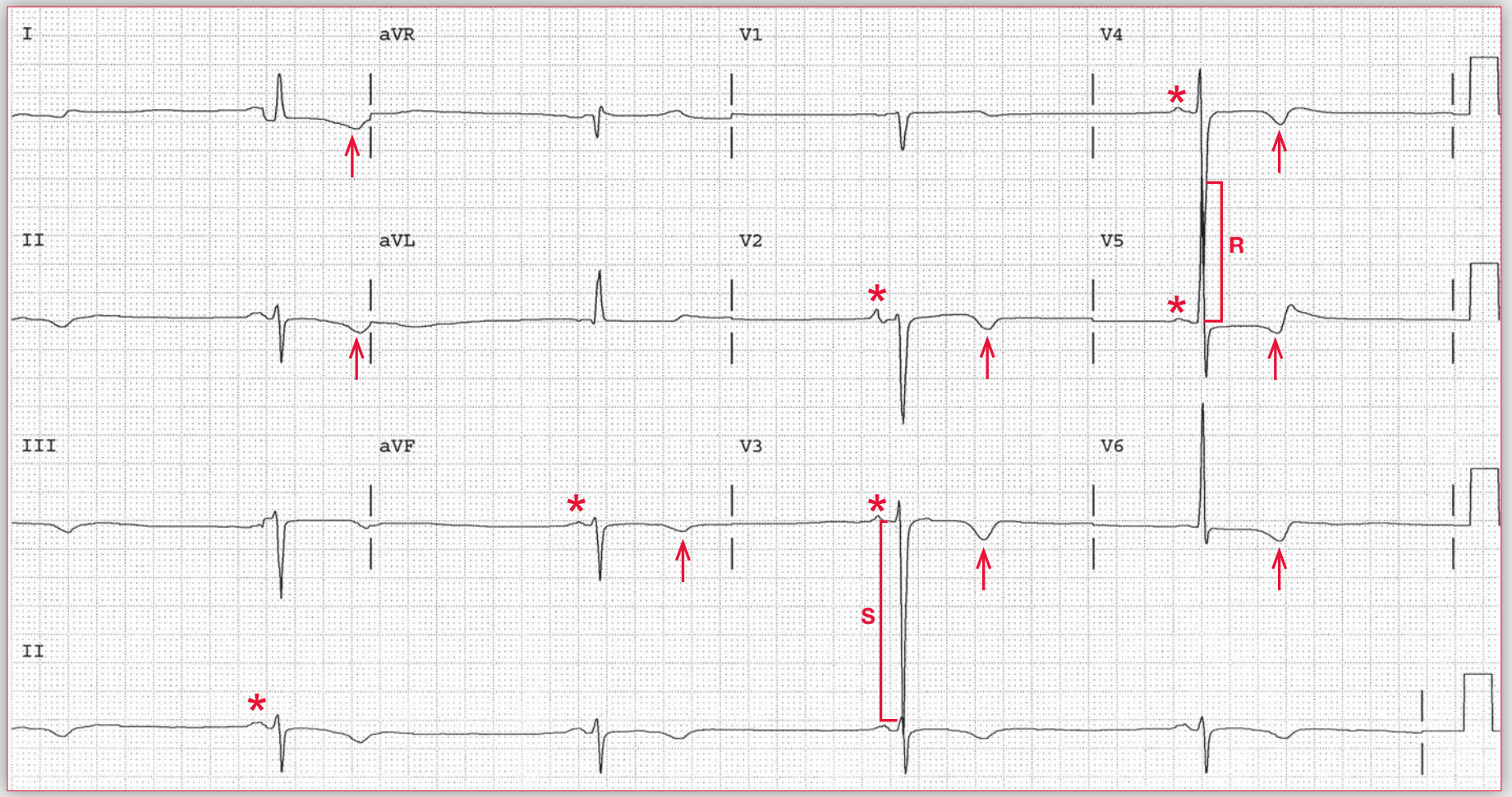
Practice ECGs

Practice Case 63

A 56-year-old woman with congenital bicuspid aortic valve with progressive symptoms of dyspnea and angina undergoes valve replacement. Her intraoperative course is uneventful and a routine postoperative ECG is obtained. The surgical team is concerned given the patient's bradyarrhythmia and consults cardiology to evaluate for permanent pacemaker placement.

Does this patient need a permanent pacemaker?





ECG 63 Analysis: Sinus bradycardia with left ventricular hypertrophy, ST-T wave changes

There is a regular rhythm at a rate of 28 bpm. There is a P wave (*), of uniform morphology, before each QRS complex, and the P wave is upright in leads I, II, aVF, and V4-V6; hence this is sinus bradycardia. The PR interval is constant (0.20 sec), the QRS complex duration is 0.10 second, and the QT/QTc intervals are normal (620/420 msec). The axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). This is a left anterior fascicular block. The other etiology for an extreme left axis is an old inferior wall myocardial infarction; in this situation there is a deep initial Q wave in leads II and aVF. The QRS complex amplitude is increased (S-wave depth in lead V3 [[ ] + R-wave amplitude in lead V5 [ ]] = 60 mm) and meets one of the criteria for left ventricular hypertrophy [LVH], *ie*, S-wave depth in lead V3 + R-wave amplitude in lead V5 ≥ 35 mm). LVH may be associated with a physiologic left axis, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). A more extreme left axis, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF), is not due to LVH but is related to a conduction abnormality, specifically a left anterior fascicular block or an old inferior wall myocardial infarction. The ST-T wave abnormalities (↑) are associated with the hypertrophy and represent chronic subendocardial ischemia. LVH and its associated ST-T wave abnormalities are often seen in severe aortic stenosis, in which myocardial remodeling results in concentric hypertrophy.

Bradyarrhythmias are common after aortic valve replacement surgery. However, they typically involve AV block due to the anatomic proximity of the aortic valve to the AV node. Persistent high-grade AV block, when associated with symptoms after an aortic valve replacement, is an indication for permanent pacing. This patient has a marked sinus bradycardia that is more commonly due to anesthetic medications used intraoperatively or an increase in vagal tone in the postoperative setting. This bradycardia will most likely resolve, so there is no indication for permanent pacing at the moment. A temporary pacemaker can be used for unstable bradycardia when associated with symptoms until the patient's sinus node recovers. ■

Notes

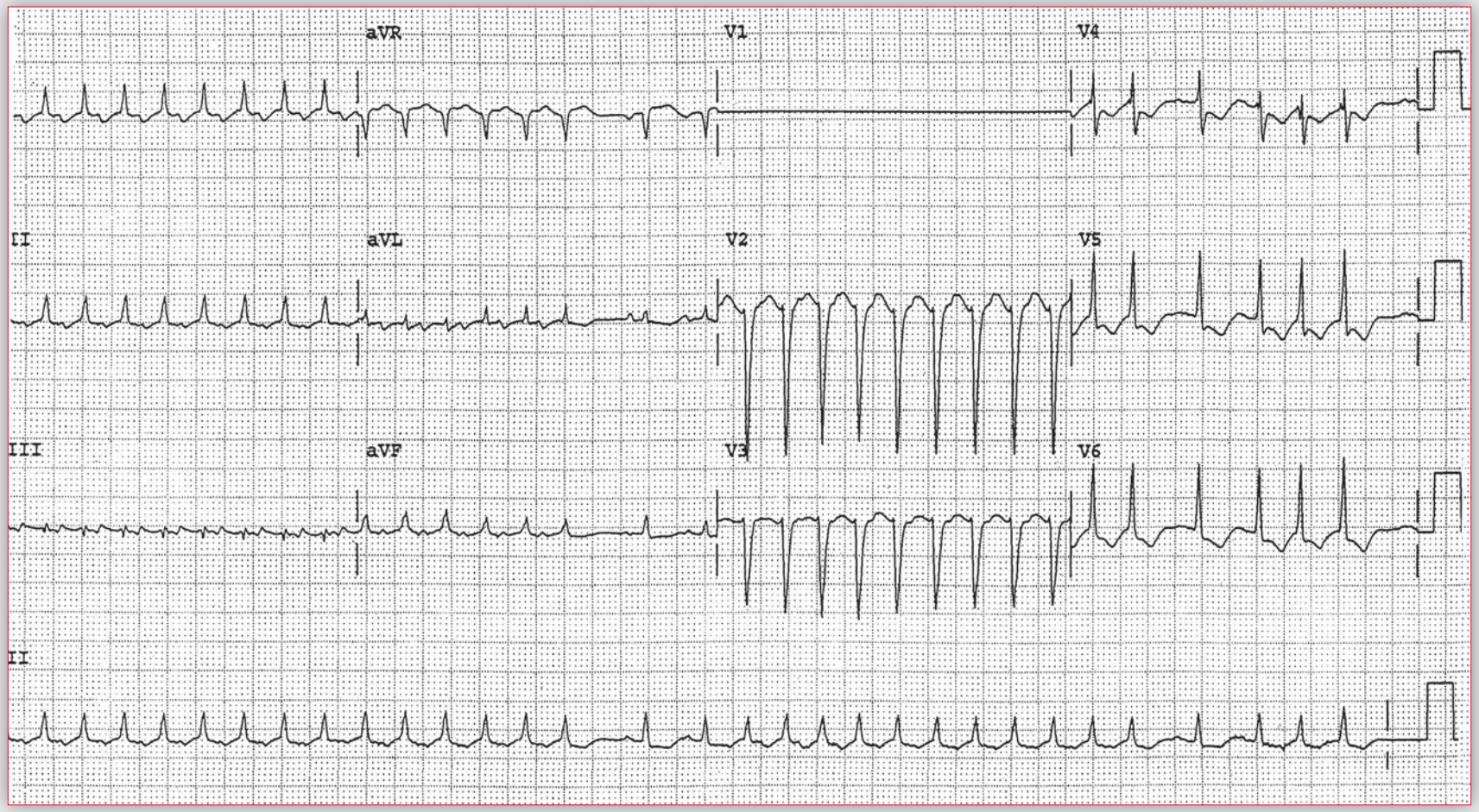
Practice Case 64

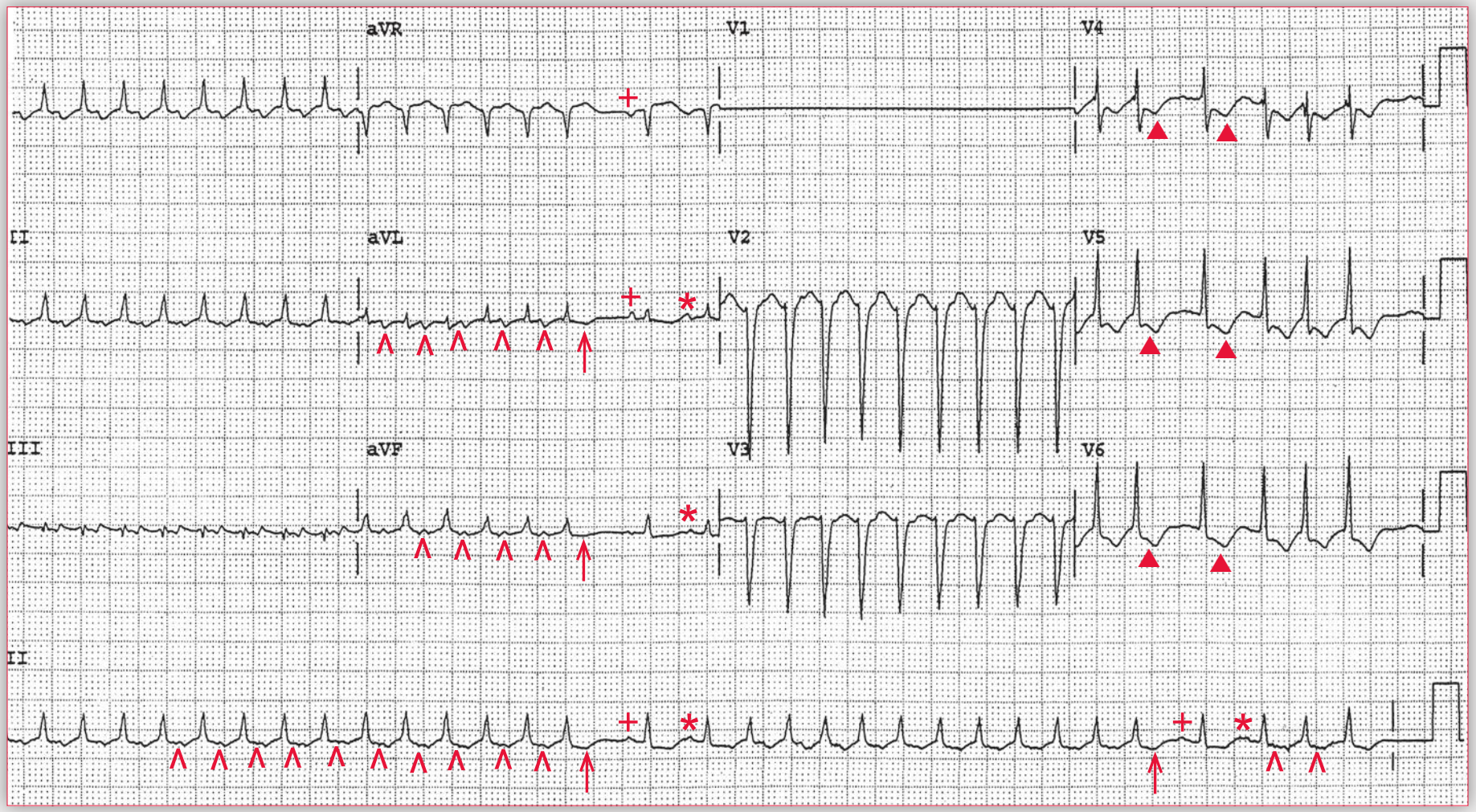
A 44-year-old man presents to a cardiologist with intermittent palpitations. He brings with him an ECG obtained during one of his recent episodes.

What does the ECG show?

What is the mechanism of the arrhythmia?

Is therapy necessary and, if so, what therapy would be effective?





ECG 64 Analysis: Atrial tachycardia with intermittent sinus beats, clockwise rotation, low-voltage limb leads, nonspecific T-wave abnormalities

The rhythm is regular at a rate of 220 bpm. There are two pauses in the rhythm, after which a distinct P wave (+) can be seen with a stable PR interval (0.14 sec). This is best seen in leads aVL and aVF as well as the lead II rhythm strip. There is a second QRS complex, thereafter with the same P wave (*) and PR interval. These are both sinus complexes. Beginning with the third QRS complex after the pause, a P wave (^) of a different morphology can be seen before the QRS complex with a PR interval of 0.20 second. As seen in leads aVL and aVF, the tachycardia ends with the absence of atrial activity (↑), after which there are two sinus beats.

This is short RP tachycardia, for which there are a number of etiologies. These include sinus tachycardia (with first-degree AV block), atrial tachycardia, ectopic junctional tachycardia, atrial flutter (often with 2:1 AV conduction), AV nodal reentrant tachycardia (slow-fast), and AV reentrant tachycardia. The fact that the P wave of the sinus complex and the tachycardia are different and the presence of a longer PR interval during the tachycardia than with the slower sinus rate exclude sinus tachycardia as the etiology. With sinus tachycardia the P waves would be identical and the PR interval during the tachycardia would be shorter as a result of a sympathetic-mediated increase in conduction velocity through the AV node. The fact that the arrhythmia terminated with the absence of a P wave excludes any arrhythmia originating or requiring the AV node as these arrhythmias generally terminate with a nonconducted P wave. This rhythm is either atrial flutter (that is slow) with 1:1 conduction or atrial tachycardia. The most likely etiology is atrial tachycardia.

The QRS complex duration is normal (0.08 sec). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (240/460 msec), although at the rapid rate accurate measurement of the QT interval is difficult. Low voltage is present in the limb leads (QRS complex amplitude < 5 mm in each lead). There is poor R-wave progression in leads V2-V3, which is due to clockwise rotation in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm. With clockwise rotation the left ventricular forces develop slowing and are seen in the lateral precordial leads. Also seen are nonspecific ST-T wave abnormalities in leads V4-V6 (▲).

Brief episodes of atrial tachycardia are benign and generally do not require therapy. If the episodes are lengthy in duration and associated with symptoms, a β-blocker or calcium-channel blocker may be effective for producing AV block and slowing the ventricular rate. A class IA, IC, or III anti-arrhythmic agent, all of which work by suppressing the atrial focus, may be used if necessary for arrhythmia suppression to provide symptom relief. A non-pharmacologic approach (*ie*, ablation of an atrial ectopic focus) can also be utilized if the patient is very symptomatic and the arrhythmia does not respond to antiarrhythmic drug therapy. ■

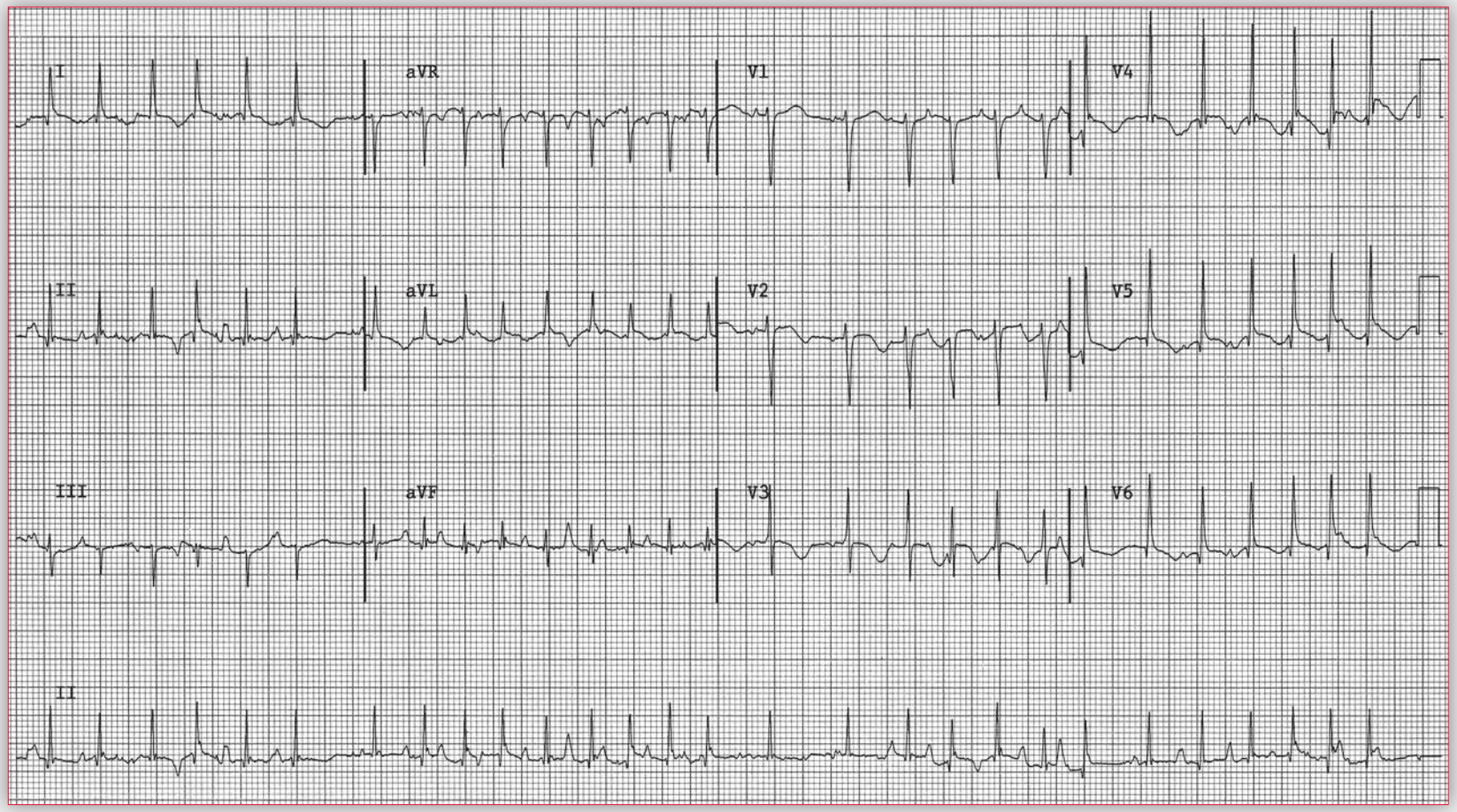
Notes

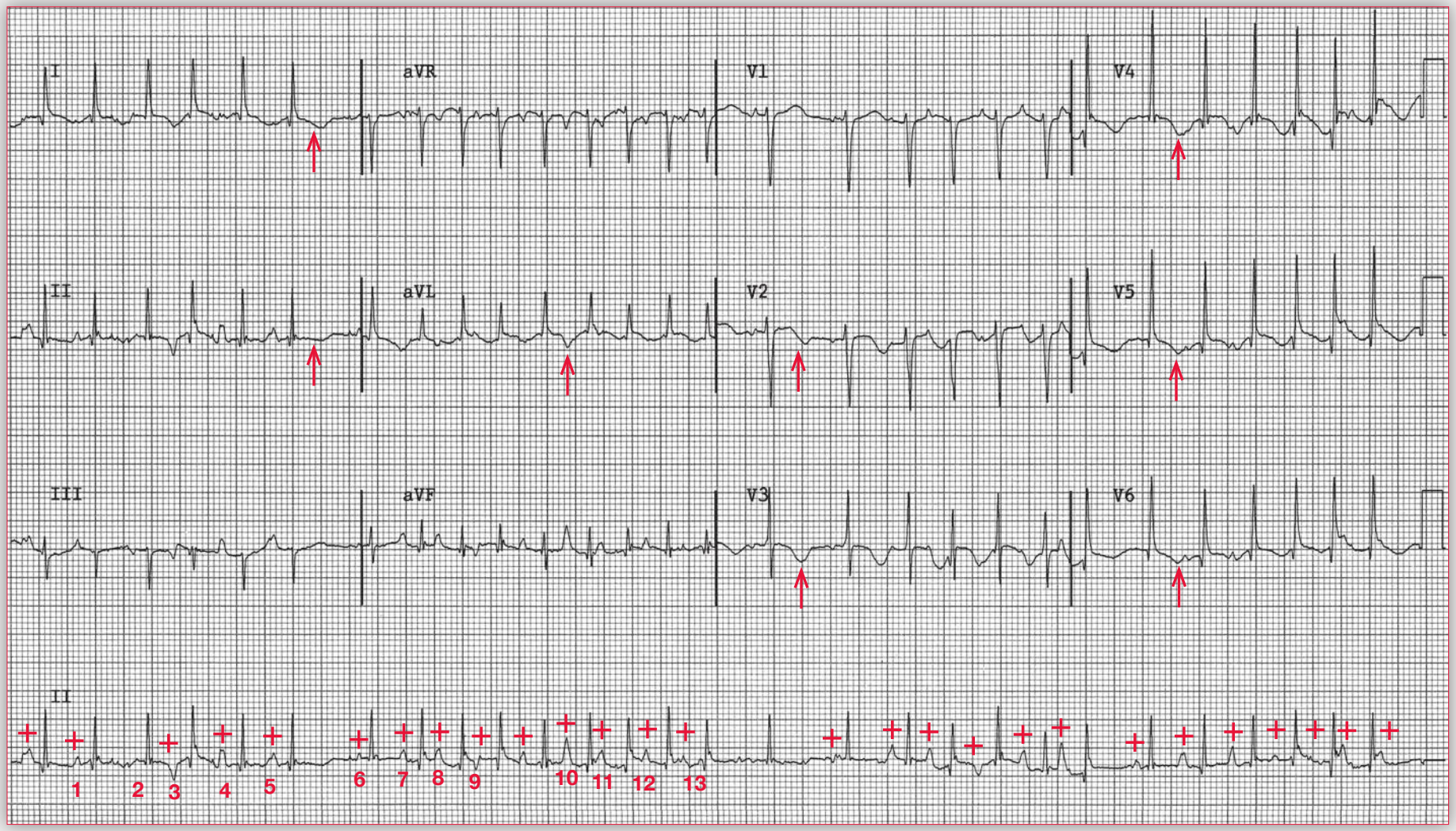
Practice Case 65

A 48-year-old woman with severe asthma is admitted to the hospital with palpitations. She is initially thought to have atrial fibrillation and is treated with attempts at rate control. Her physicians avoid β-blockers because she was observed in the past to have worsening bronchospasm with metoprolol. Instead, they treat her with digoxin but have very little success in controlling the arrhythmia.

What does her ECG show?

What other therapies should be tried?





ECG 65 Analysis: Multifocal atrial tachycardia, nonspecific ST-T wave changes

The rhythm is irregularly irregular at an average rate of 168 bpm. There are only three supraventricular rhythms that are irregularly irregular:

- Sinus arrhythmia, in which there is only one P-wave morphology and a stable PR interval
- Multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm), in which there are three or more P-wave morphologies and often PR intervals without any one P-wave morphology being dominant
- Atrial fibrillation in which there are no organized P waves

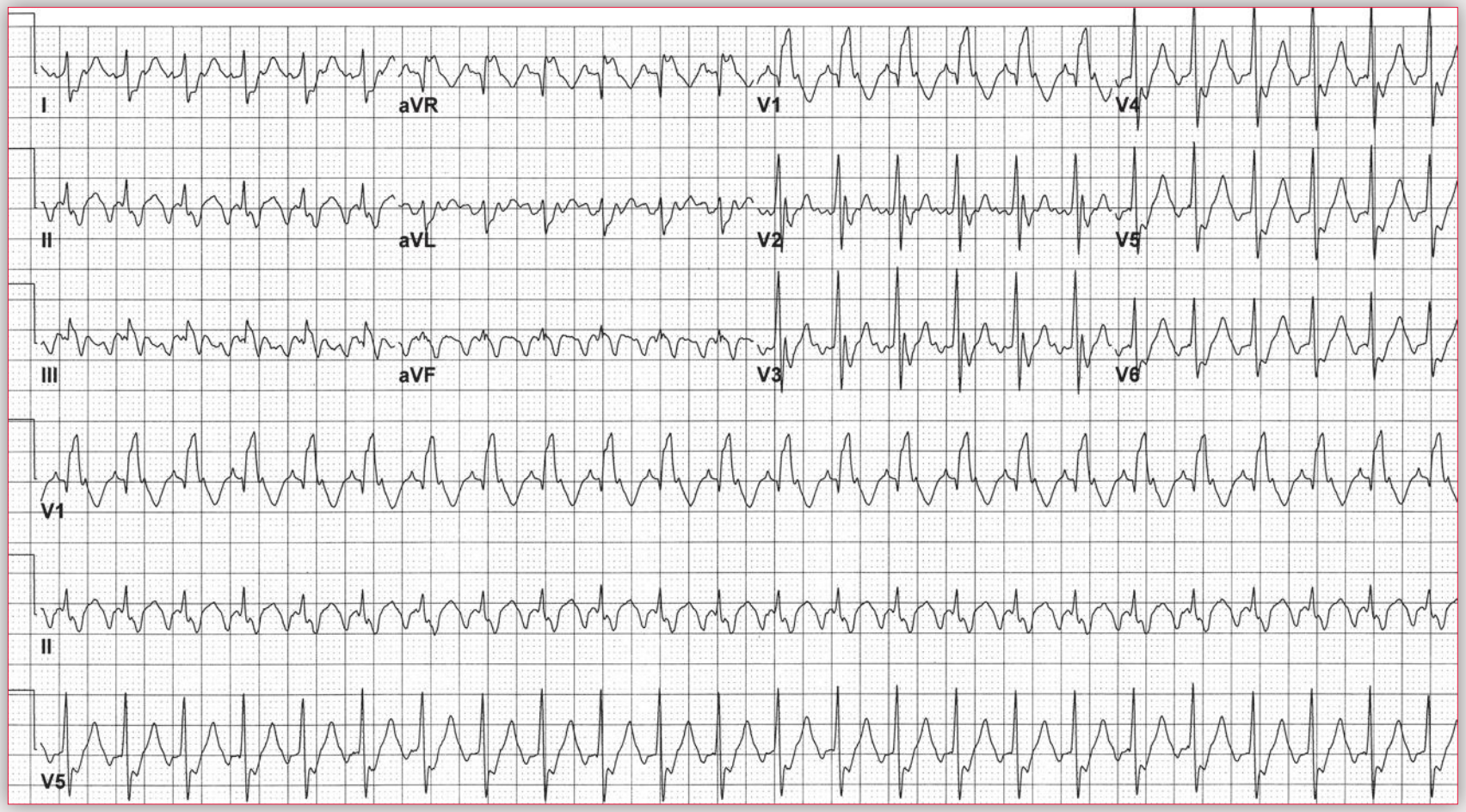
There is a P wave (+) before each QRS complex, but there are multiple (three or more) P-wave morphologies with no P-wave morphology being dominant. Therefore, this is multifocal atrial tachycardia and not atrial fibrillation. The QRS complexes are of normal duration (0.08 sec) and morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (260/440 msec). There are diffuse ST-T wave changes (↑) noted.

Multifocal atrial tachycardia is a difficult arrhythmia to treat. Initial treatment is directed at the underlying medical condition, in this case the severe bronchospasm. Initial therapy for the arrhythmia itself would be rate control. It would be reasonable to treat with a calcium-channel blocker, which may reduce AV nodal conduction and thus slow the ventricular rate. There is some evidence that a calcium-channel blocker may depress the multiple ectopic foci and hence terminate the arrhythmia. In some cases multifocal atrial tachycardia will revert to atrial fibrillation. ■

Practice Case 66

You are supervising an outpatient adenosine-based cardiac stress test on a 55-year-old woman with diabetes and familial premature coronary disease. Test indications include exertional and non-exertional palpitations and breathlessness. During the initial portion of adenosine infusion, her heart rate

ECG 66A

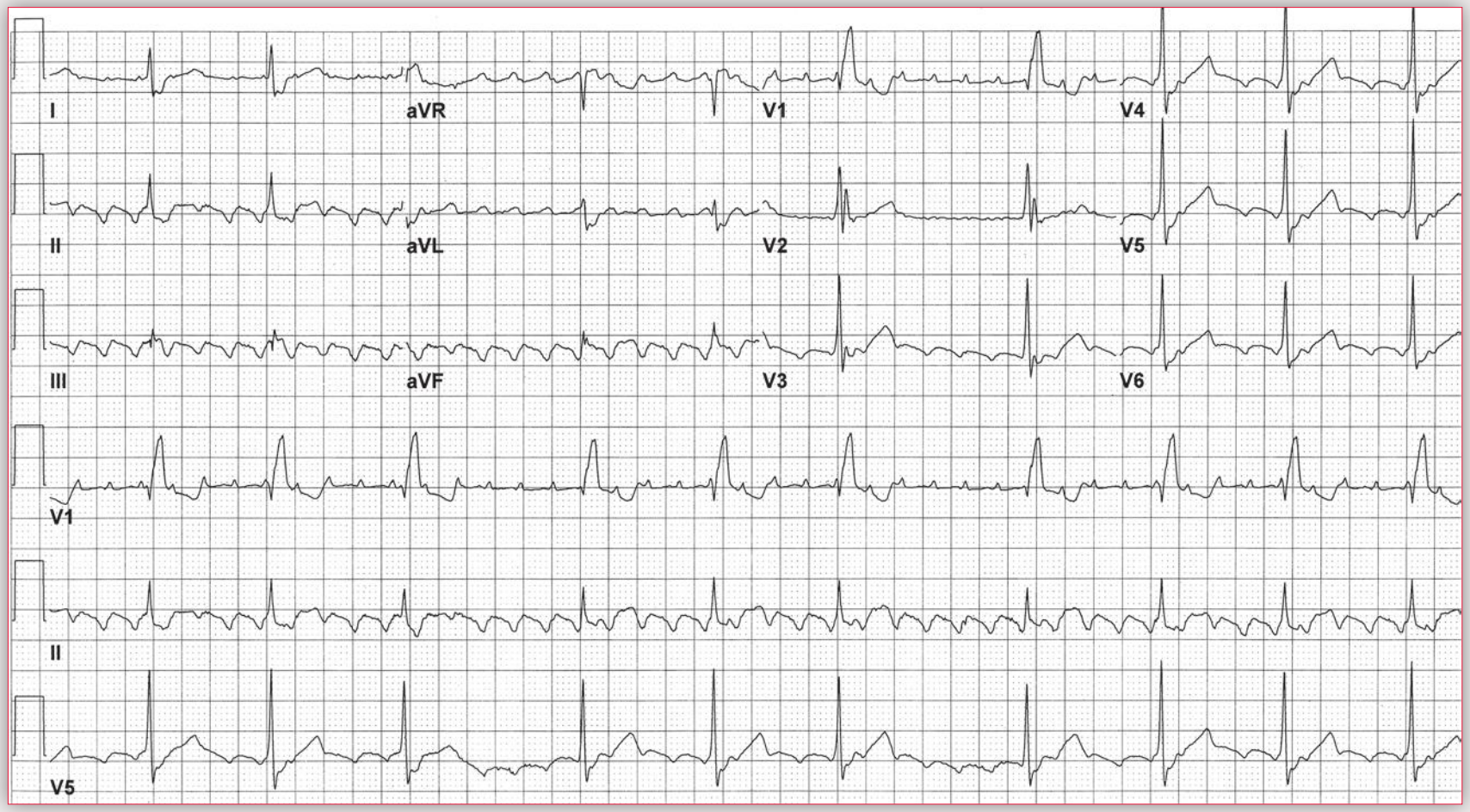


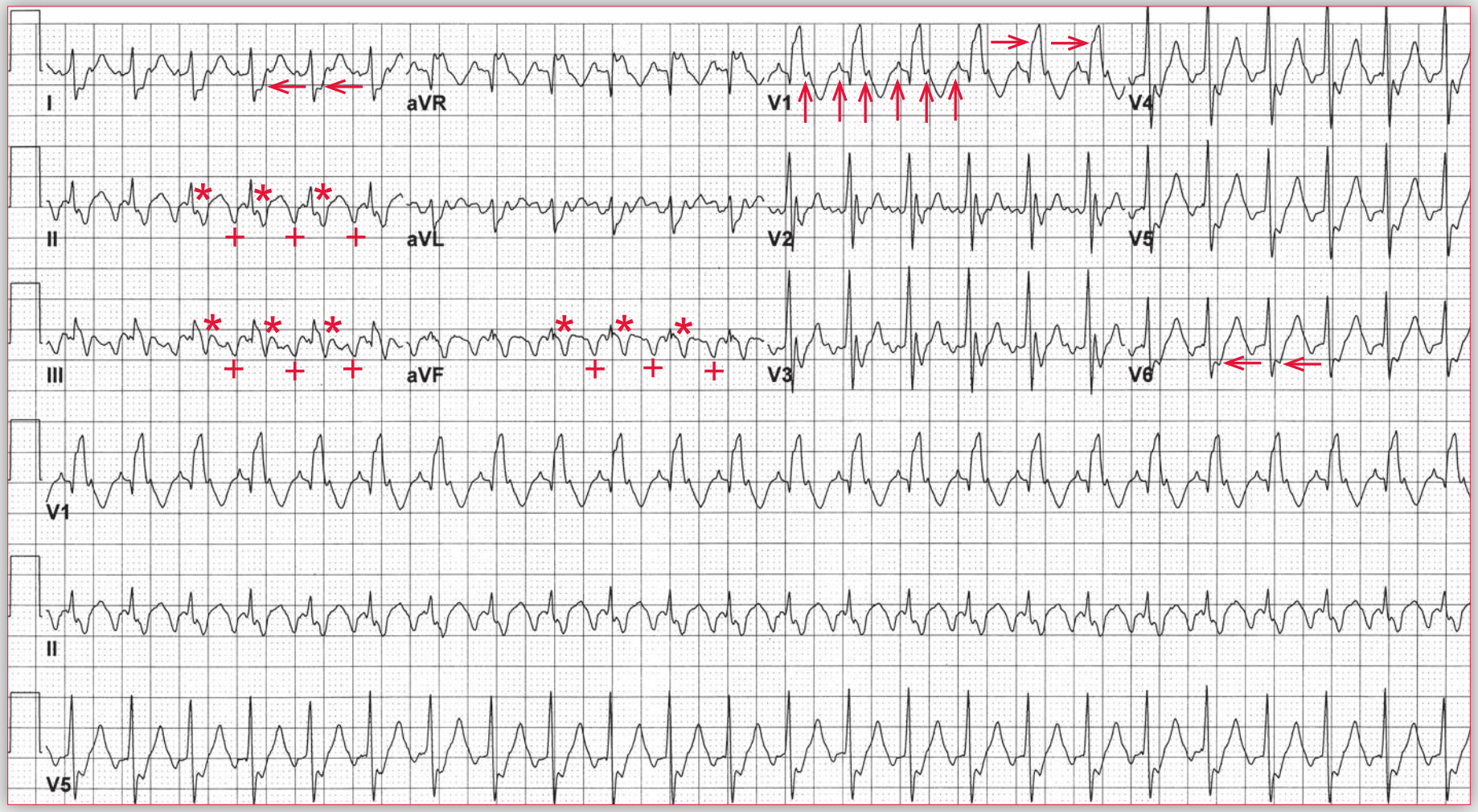
abruptly increases and her symptoms are reproduced. Shortly thereafter, her heart rate abruptly slows. The exercise technician looks at the ECGs and states, "Well, it looks like she has atrial fibrillation!" You review the ECGs obtained during (ECG 66A) and after (ECG 66B) her episode of tachycardia.

Do you agree with the technician's assessment?

If not, what is your diagnosis?

ECG 66B





ECG 66A Analysis: Atrial flutter with 2:1 conduction, right bundle branch block

The rhythm in **ECG 66A** is regular at a rate of 150 bpm. The QRS complex is widened (0.14 sec), and there is an RSR′ morphology in lead V1 (→) and a broad S wave in leads I and V5-V6 (←); this is right bundle branch block. The axis is normal (positive QRS complex in leads I and aVF, *ie*, between 0° and +90°). The QT/QTc intervals are normal (280/440 msec and 220/350 msec when corrected for the prolonged QRS duration). A negative atrial waveform can be seen before each QRS complex in leads II, III, and aVF (+). An identical atrial waveform can be seen at the end of the QRS complex in these leads (*). In addition, a distinct atrial waveform before and after each QRS complex can be seen in lead V1 (↑). The interval between these waveforms is constant, and the atrial rate is 300 bpm. There is no isoelectric baseline between these waveforms; rather the baseline is constantly undulating (saw-tooth pattern). Hence this is atrial flutter with 2:1 AV conduction.

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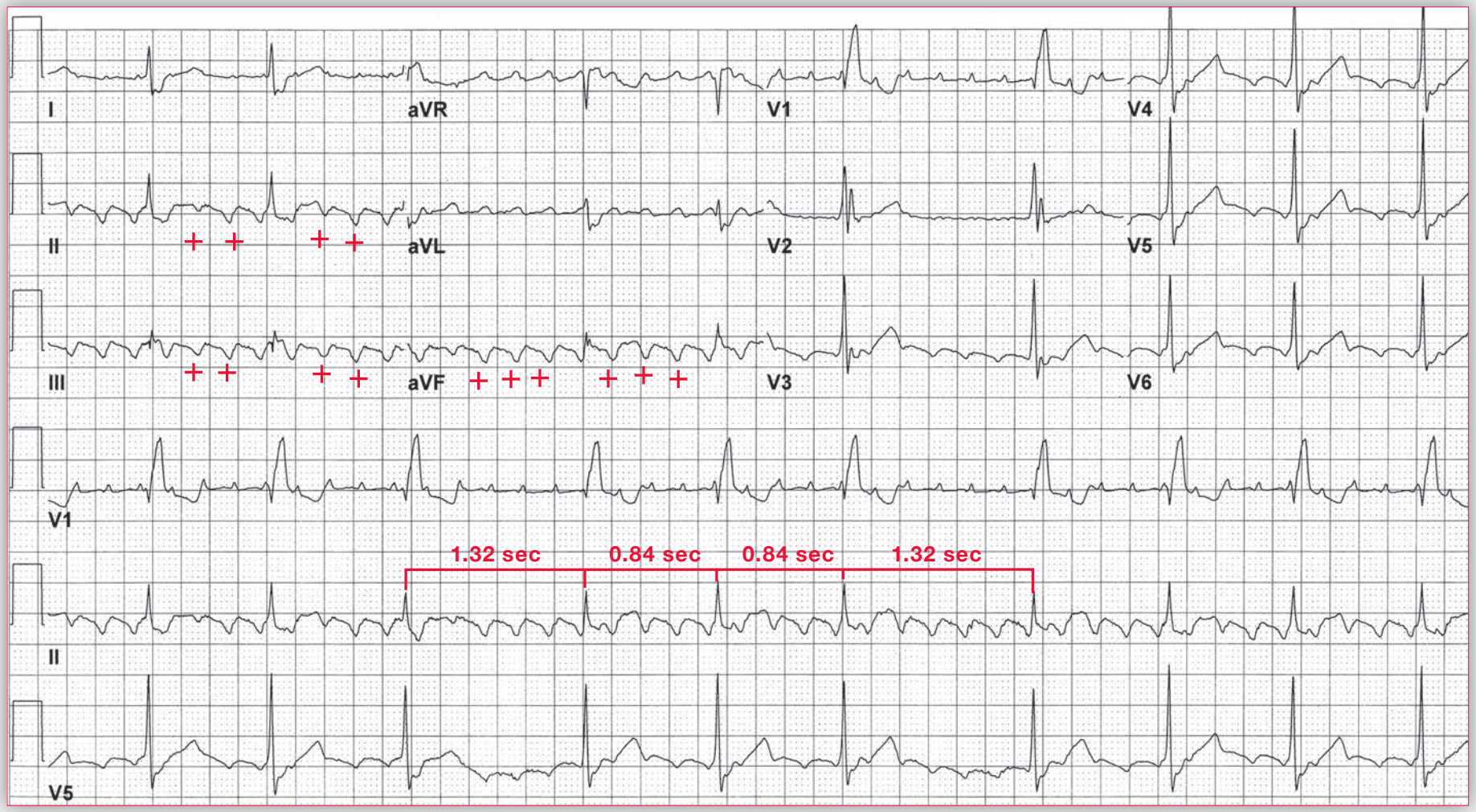


ECG 66B Analysis: Right bundle branch block, counterclockwise rotation, type I atrial flutter with variable AV block

ECG 66B was obtained during infusion of adenosine, an AV nodal blocking agent. The QRS complex duration, morphology, and axis are identical to what is seen in **ECG 66A**. The QT/QTc intervals are also the same as in **ECG 66A**. The ventricular rate is now 60 bpm and the rhythm is irregular; however, all of the short intervals are identical to each other (0.84 sec) and the two longer intervals (1.32 sec) are also the same. Hence the rhythm is regularly irregular. As a result of the increased AV block, the atrial flutter waveforms are now apparent (**+**). The atrial rate is 300 bpm, identical to that seen in **ECG 66A**. The atrial waveforms have a typical saw-tooth appearance, and there is no isoelectric baseline between them as noted in leads II, III, and aVF. It can be seen that there is a variable degree of AV block present (4:1 and 6:1), accounting for the irregularity. Also noted are differences in the intervals between the flutter wave and QRS complex, which is the result of antegrade concealed conduction. In this situation, some of the atrial impulses travel through the AV node to activate the ventricles, some are blocked within the AV node, and some may only partially penetrate and partially depolarize the AV node but not get through (*ie*, they are concealed). However, the partial depolarization and refractoriness of the AV node results in slowing of AV nodal conduction of the subsequent impulse.

Atrial flutter is not a serious arrhythmia, but it often causes symptoms as the result the rapid ventricular rate. In this patient the onset of the arrhythmia replicated the symptoms of palpitations and breathlessness. Therefore, it is unlikely that ischemia is the cause and hence there is no reason for further testing. Therapy should be directed at controlling the symptoms. Initial therapy is rate control when the arrhythmia occurs. However, given the episodic nature of the arrhythmia, arrhythmia suppression would be appropriate. This can be achieved with the use of a class IA, IC, or III anti-arrhythmic drug or a non-pharmacologic approach, such as radiofrequency ablation. ■

Notes

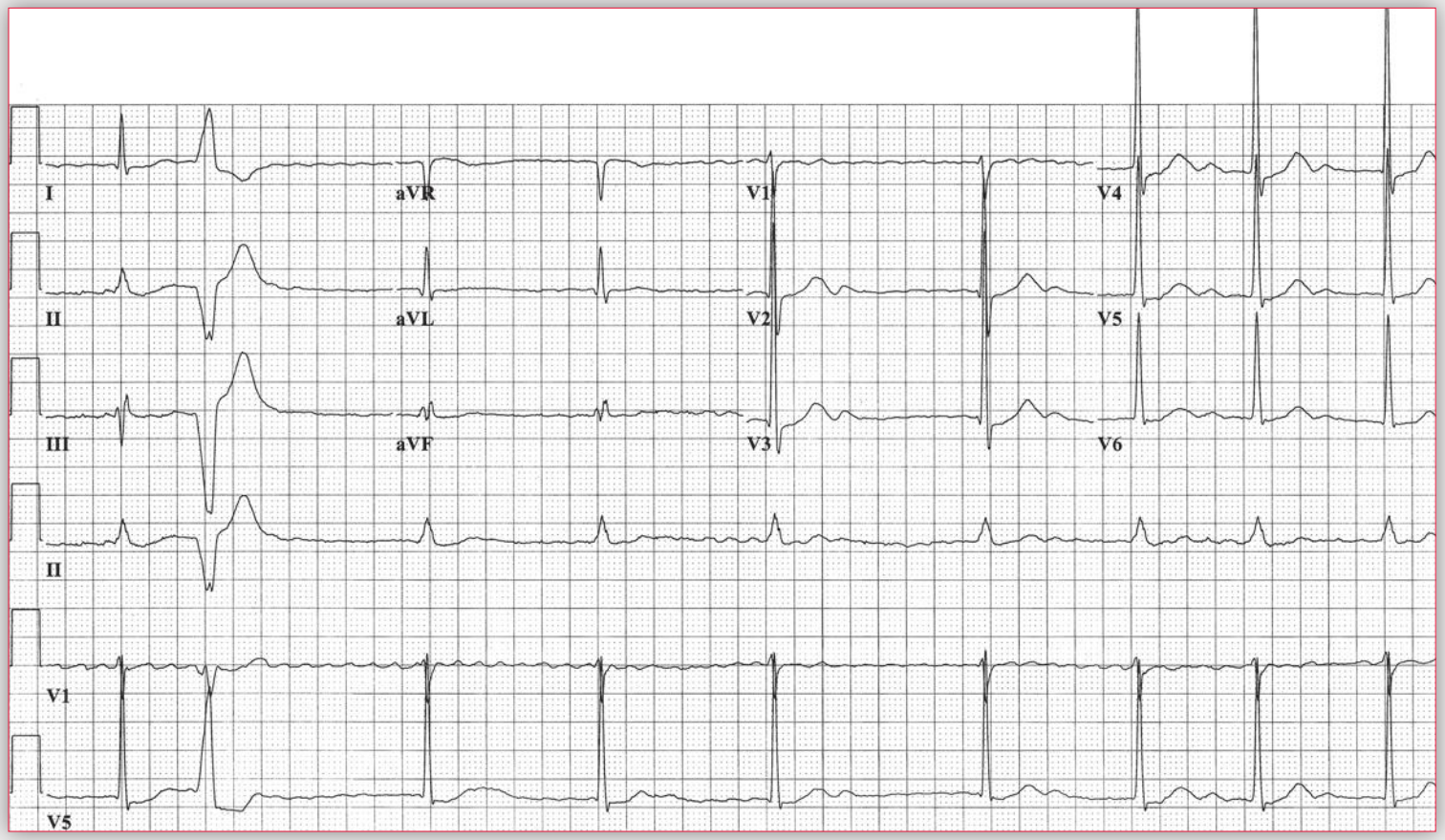
Practice Case 67

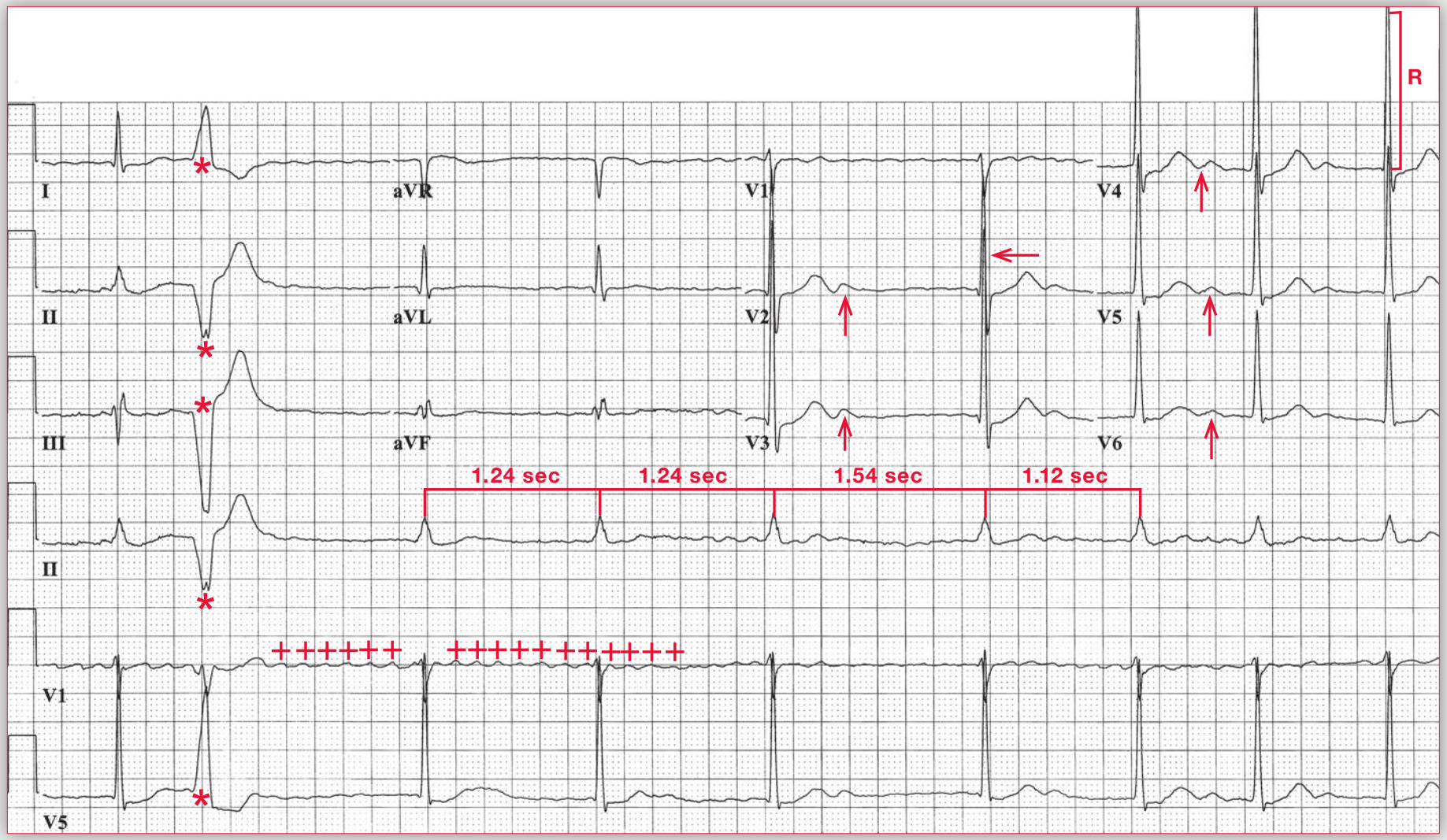
A 68-year-old woman with a known history of paroxysmal atrial fibrillation on digoxin therapy and uncontrolled hypertension presents to her primary care physician (PCP) for follow-up of her hypertension. Her blood pressure continues to be mildly elevated despite calcium-channel blocker therapy, and she is started on hydrochlorothiazide. Per her usual state, she is in sinus rhythm. One week later the woman calls her PCP with complaints of fatigue, breathlessness with minimal exertion, and muscle aches. She has also noted an irregular pulse. She denies angina. She is brought into the urgent care clinic, where her physical exam is notable for a pulse of 54 bpm, and her blood pressure and cardiopulmonary exam are normal. An ECG is obtained.

What are the findings on the ECG?

Do they explain her symptoms?

What is the etiology of these findings?





ECG 67 Analysis: Atrial fibrillation with slow ventricular response, counterclockwise rotation, U waves, premature ventricular complex, left ventricular hypertrophy

The rhythm is irregularly irregular at a rate of 54 bpm. There are no obvious organized P waves; rather, there are fine and rapid waveforms (+) that are irregular in amplitude, morphology, and interval. These are atrial fibrillatory waves and the underlying rhythm is atrial fibrillation with a slow ventricular response. A single premature ventricular complex can be seen (second QRS complex [*]).

The QRS complex axis, duration (0.10 sec), and morphology are normal. However, there is early transition (*ie*, a tall R wave in lead V2 [←]) as a result of counterclockwise rotation in the horizontal plane. The axis in the horizontal plane is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation the left ventricular forces are apparent early in the precordial leads (*ie*, a tall R wave in lead V2). The amplitude of the QRS complex is tall (R-wave amplitude in leads V3-V4 = 33 mm [ ]]), which is diagnostic for left ventricular hypertrophy (*ie*, S-wave or R-wave in any precordial lead ≥25 mm). The QT/QTc intervals are normal (460/440 msec).

In addition, prominent U waves are seen in leads V2-V6 (↑). The U wave, which follows the T wave, represents late repolarization and is believed to be the result of repolarization of the His-Purkinje system, which is the last part of the myocardium to become repolarized. It is often seen as a low-amplitude, positive waveform in the right precordial leads (V1-V3) and it often becomes more prominent with bradycardia. However, an increase in the amplitude of the U wave or its presence in the lateral precordial leads (V4-V6) is suggestive of hypokalemia.

The slow ventricular response rate noted in this patient is the result of combined therapy with two AV nodal blocking agents. Digoxin slows AV nodal conduction as a result of increased vagal tone. Calcium-channel blockers have a direct effect on AV nodal conduction properties because the AV nodal electrophysiologic properties are mediated by a slow calcium current.

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It has been observed that in patients with a history of paroxysmal or intermittent atrial fibrillation, the use of digoxin is associated with increased episodes of atrial fibrillation and may even precipitate sustained atrial fibrillation. This is the result of digoxin's vagal effect. The effect of increased parasympathetic tone on the atrial myocardium is a shortening of the atrial myocardium refractory period. However, as vagal innervation of the atrial myocardium is very heterogeneous, digoxin-induced enhancement of vagal tone increases heterogeneity of myocardial electrophysiologic properties, which is a precondition that can increase the potential for atrial fibrillation. Therefore, digoxin is not appropriate therapy for prevention of paroxysmal atrial fibrillation.

Although prominent U waves may be seen with bradycardia, the presence of prominent U waves in leads V2-V6 suggests hypokalemia, which if present is likely the result of the addition of hydrochlorothiazide. This is a thiazide diuretic, and one of the most common side effects of this class of drugs is hypokalemia. The presence of hypokalemia may result in an increase in digoxin effect and possible digoxin toxicity, even with stable and therapeutic digoxin levels. It is likely that

her symptoms of fatigue and muscle aches are the result of hypokalemia. The shortness of breath as well as fatigue may be related to atrial fibrillation, especially with the slow ventricular response rate. In patients with hypertension and possible left ventricular hypertrophy the development of atrial fibrillation and the loss of atrial contraction could result in significant hemodynamic consequences, including the symptoms experienced by this patient.

Initial workup should include measurement of serum electrolytes and correction of any imbalances, particularly hypokalemia. There is also a possibility that hydrochlorothiazide caused dehydration and resultant pre-renal azotemia. This could be associated with an unexpected increase in digoxin levels. Along with hypokalemia this could result in digoxin toxicity. It would also be appropriate to withhold digoxin and verapamil therapy to see if her heart rate increases and her symptoms resolve. ■

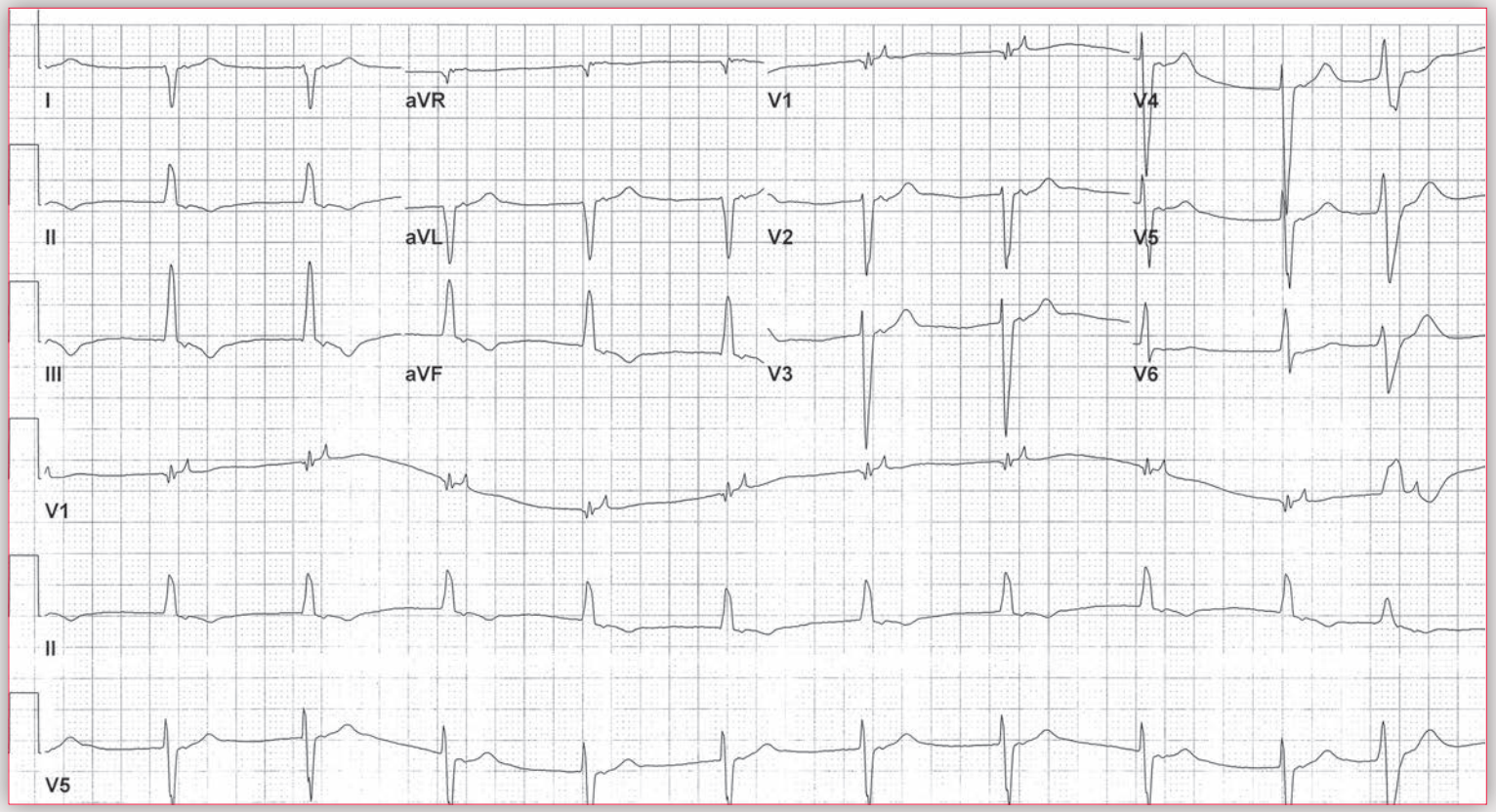
Practice Case 68

A 44-year-old man who recently emigrated from Brazil presents as a new patient to his primary doctor. Although he has no acute complaints, he notes a slow but progressive decline in his ability to perform physical tasks. Specifically, he becomes more easily fatigued with daily tasks such as running errands. He denies anginal symptoms, weight changes, extremity edema, orthopnea, palpitations, or pre-syncopal symptoms. A review for gross and occult bleeding is negative.

He is unaware of his medical history, except for one illness as a teenager that resulted in hospitalization with months of recuperation. He does not recall the diagnosis but remembers debilitating chest and abdominal pain with fevers. He has been healthy throughout his adult life and has not had any surgeries.

He is not taking any medications. Members of his immediate family are all alive and well. His social history is remarkable only for his country of origin.

ECG 68A



Practice Case 68

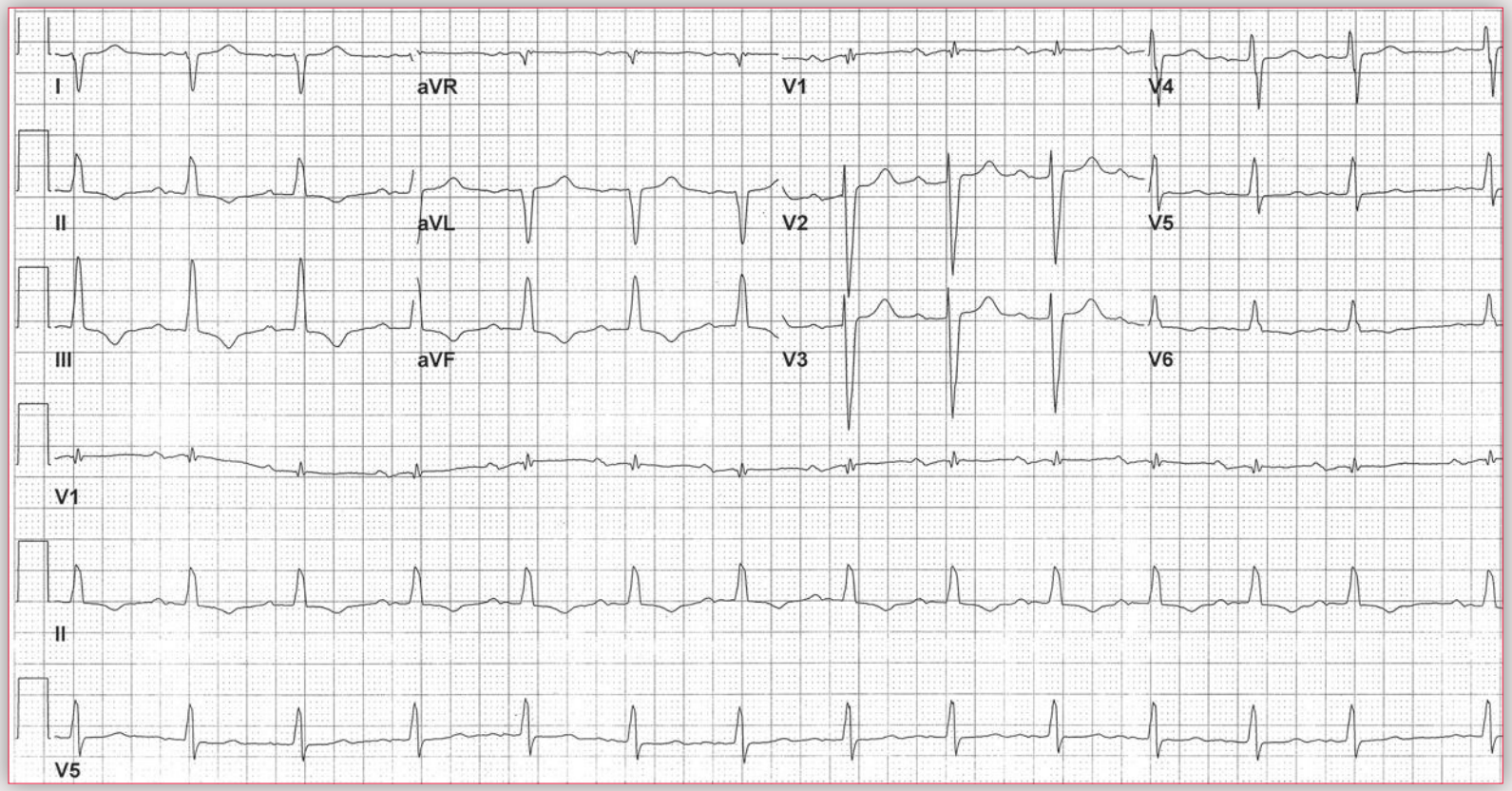
His physical exam is unremarkable. Vital signs are within normal limits. No rashes are noted. Lungs are clear on auscultation. His jugular venous pulse is normal, although prominent but regular pulsations are present. They appear to occur after the carotid artery upstroke. The cardiac exam displays normal point of maximal impulse without heaves and normal S1 and S2 without murmurs or rubs. His abdominal, extremity, and neurologic exams are normal.

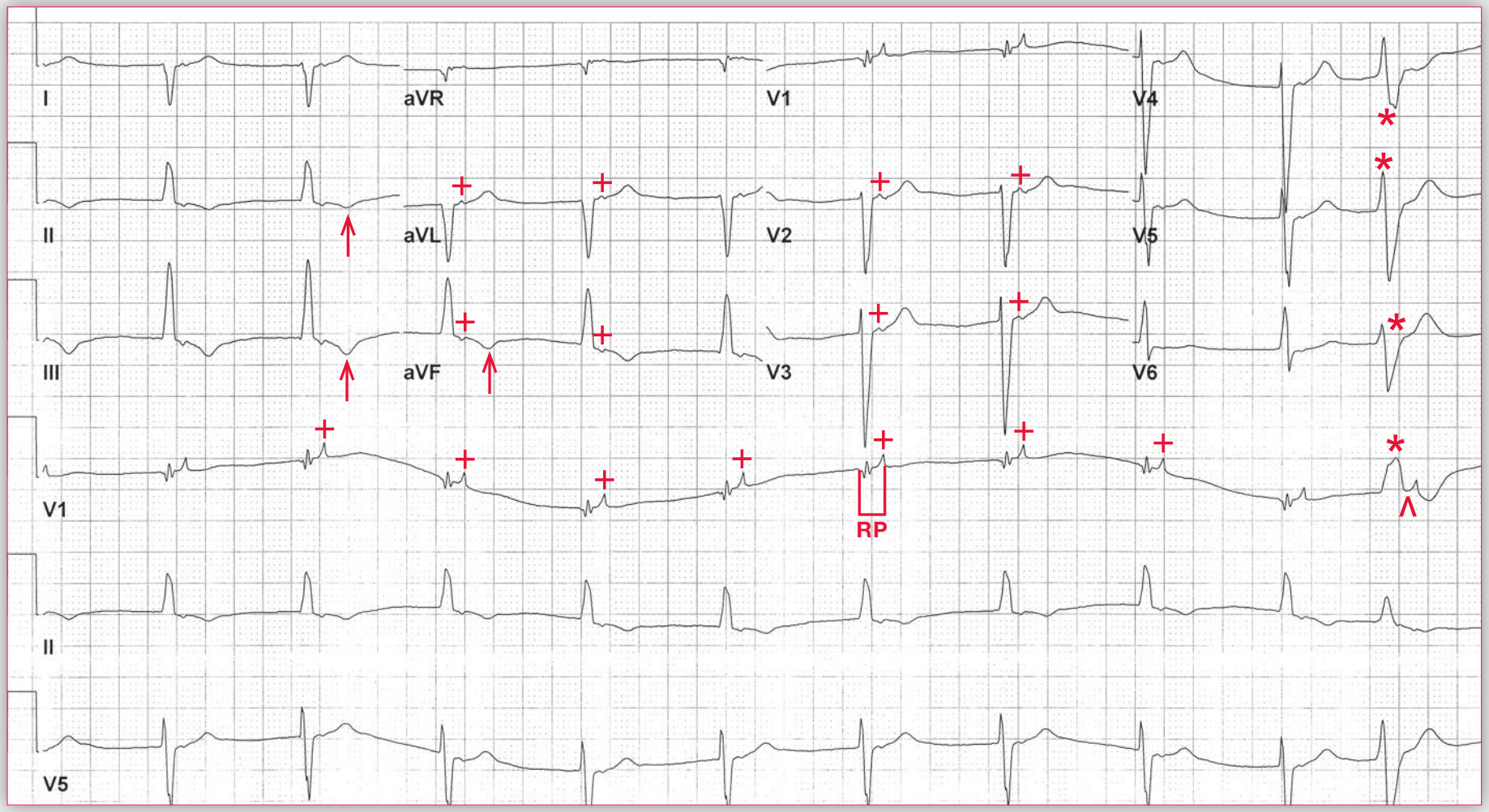
An ECG (68A) is obtained as part of his routine evaluation. When he returns for follow-up 2 weeks later, a repeat ECG (68B) is obtained.

What abnormalities are noted?

What diagnosis is suggested in ECG 68A, particularly when compared with ECG 68B?

ECG 68B





ECG 68A Analysis: Junctional rhythm with retrograde (ventriculoatrial) conduction, left posterior hemiblock, nonspecific ST-T wave abnormalities

ECG 68A shows a regular rhythm at a rate of 64 bpm. The QRS complexes have a normal duration (0.10 sec) and morphology. The QT/QTc intervals are normal (400/410 msec). However, the axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). In the absence of other reasons for the right axis, the etiology is left posterior fascicular block (*ie*, block in the left posterior fascicle of the left bundle). The left bundle divides into two major fascicles that activate the left ventricle (*ie*, the left anterior and left posterior fascicles). When left posterior fascicular block is present, all left ventricular activation originates from the left anterior fascicle and is directed down and to the right. Hence fascicular block results in a shift in the electrical axis in the frontal plane. As left ventricular activation still occurs via the normal Purkinje pathway, the QRS complex width is not prolonged with fascicular block; the only change is an axis shift.

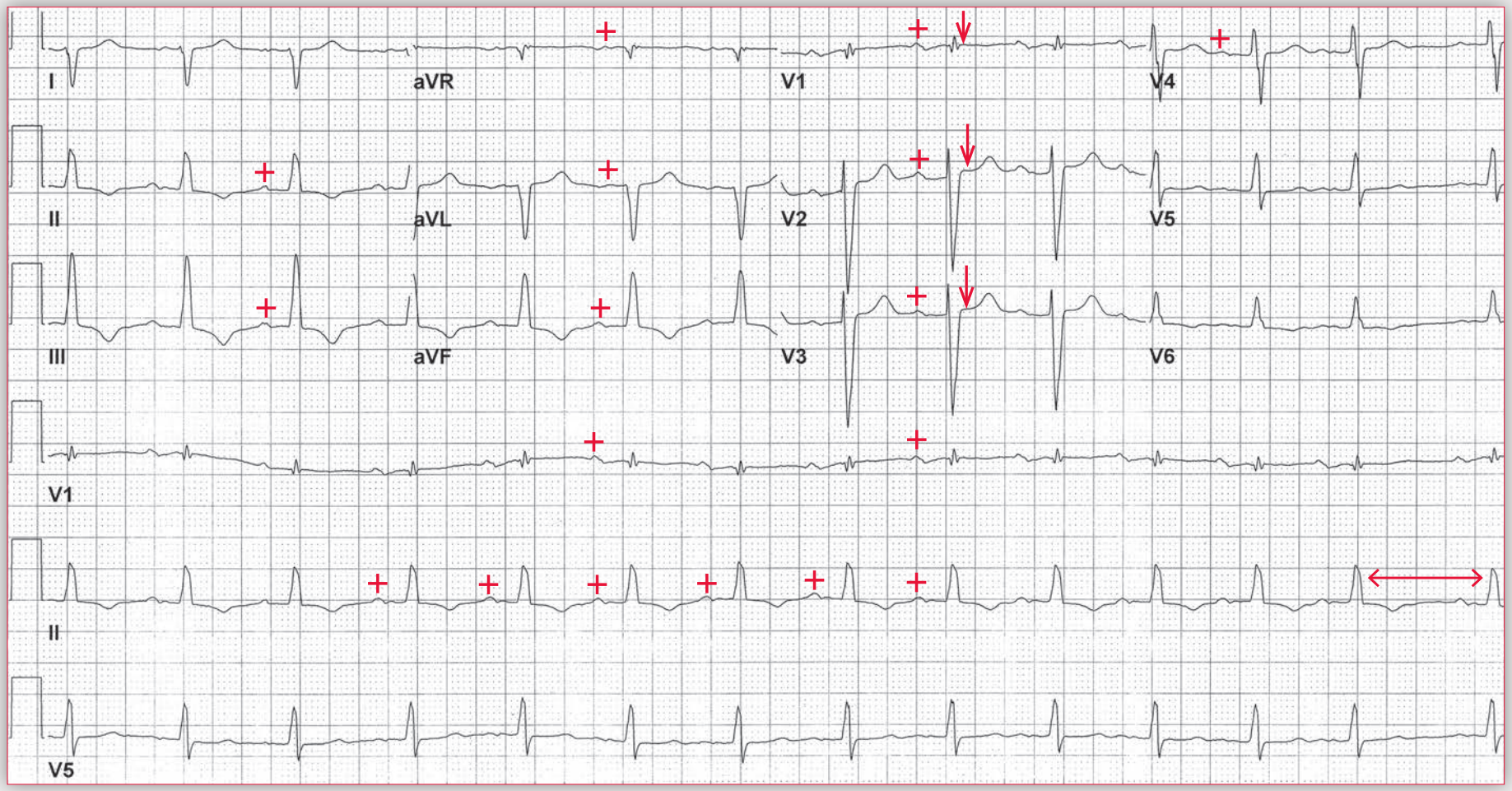
Because left posterior fascicular block is a diagnosis of exclusion, other causes for a right axis must be excluded, such as a lateral wall infarction pattern (an initial Q wave in leads I and aVL), right ventricular hypertrophy (associated with a tall R wave in lead V1 and often right atrial abnormality or hypertrophy), right–left arm lead switch (negative P waves in leads I and aVL), dextrocardia (in which there are also inverted P waves in leads I and aVL and reverse R-wave progression across the precordium), and Wolff-Parkinson-White pattern (short PR interval and delta wave).

No P waves are seen before any of the QRS complexes. As the QRS complexes are normal in duration, this is a junctional rhythm. However, there are P waves seen after the QRS complex (presenting as a bump or notching within the ST segment) (+). The ST segment should be smooth, and any notches or bumps suggest a superimposed P wave. The P waves are negative in leads II, aVF, and V2-V5. These are "retrograde" P waves and represent retrograde atrial activation due to ventriculoatrial conduction from a junctional complex. The RP interval is constant (↔). Hence this is a junctional rhythm with retrograde P waves.

A junctional rhythm is identified by a regular rhythm with a supraventricular QRS complex morphology similar to that of the sinus complex. There are no P waves in front of the QRS complexes, but an inverted (retrograde) P wave may be present following the QRS complex (due to ventriculoatrial conduction) with a stable RP interval.

The last QRS complex (*) is premature and wider (0.16 sec), has no a preceding P wave, and has an abnormal morphology. This is a premature ventricular complex. A retrograde P wave (^) is seen after this premature complex. In addition, the T waves are negative (inverted) (↑) in leads II, III, and aVF; this is a nonspecific change.

continues



ECG 68B Analysis: Sinus rhythm with first-degree AV block, nonspecific ST-T wave abnormalities, sinus node pause due to sinus node arrest

ECG 68B shows a regular rhythm at a rate of 80 bpm. The QRS complexes are identical to those in **ECG 68A**. The QT/QTc intervals are normal (380/440 msec). However, a P wave (**+**) can be seen before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm with first-degree AV block (or prolonged AV conduction). Note that there are no notches or bumps after the QRS complex (↓) or within the ST segment, confirming that this was a retrograde P wave, as now the rhythm is no longer junctional but sinus. There is one long RR interval notable at the end of the ECG (↔). No P wave is seen during this pause. Thus this represents a sinus pause. There are two etiologies for a sinus pause: (1) a sinus node exit block in which the PP interval around the pause is equal to two sinus intervals or (2) sinus node arrest in which the PP interval around the pause is unrelated to the sinus PP interval. In this case, the PP interval around the pause is less than two sinus intervals and this is a sinus node arrest.

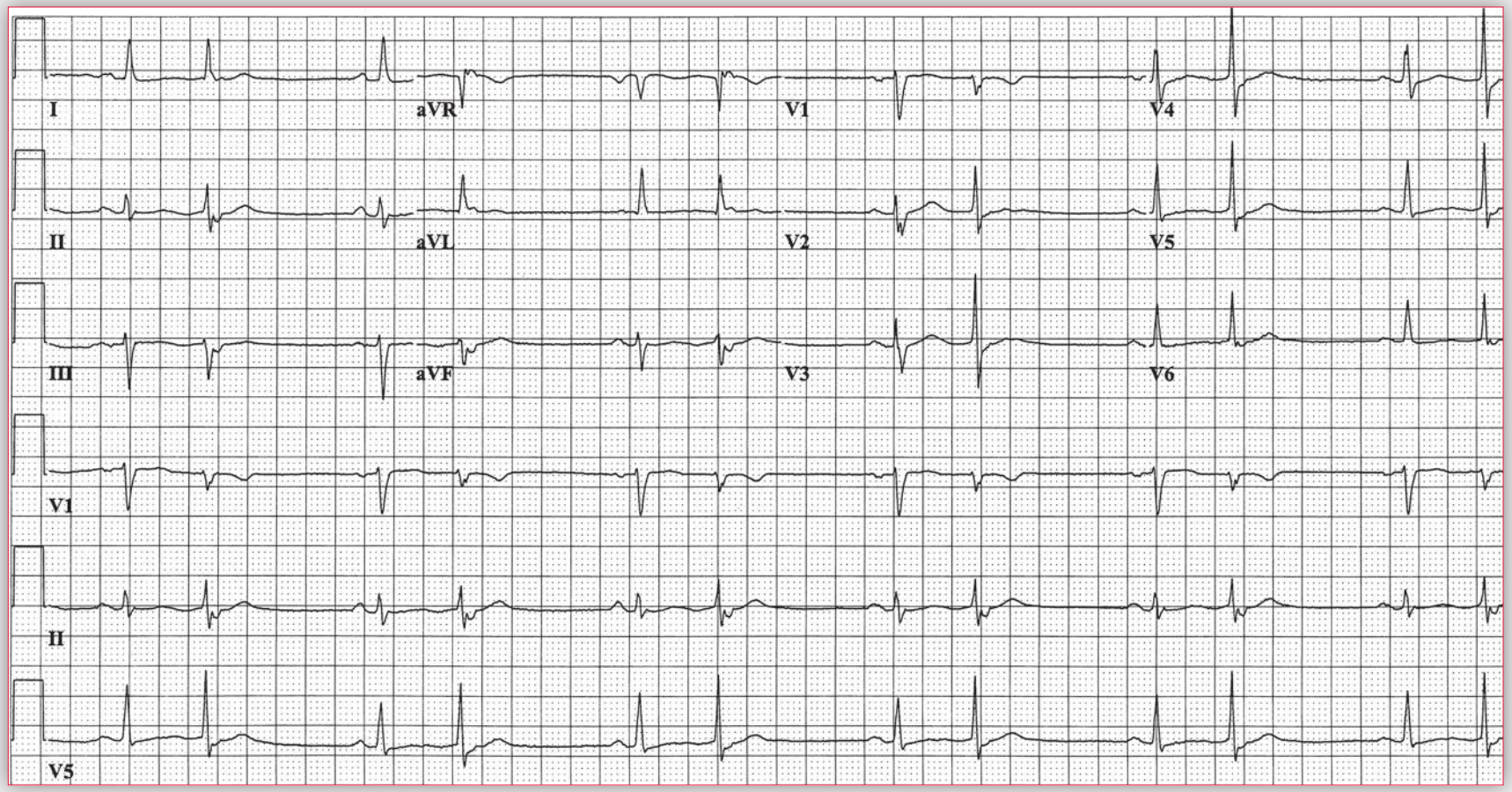
In a patient from a region of the world endemic to *Trypanosoma cruzi* with a history of a notable childhood febrile illness and current ECG features of cardiomyopathy, Chagas disease should be suspected. Even though the physical exam is not consistent with depressed left ventricular function or volume overload, echocardiography and serologic testing for *T. cruzi* are indicated. ■

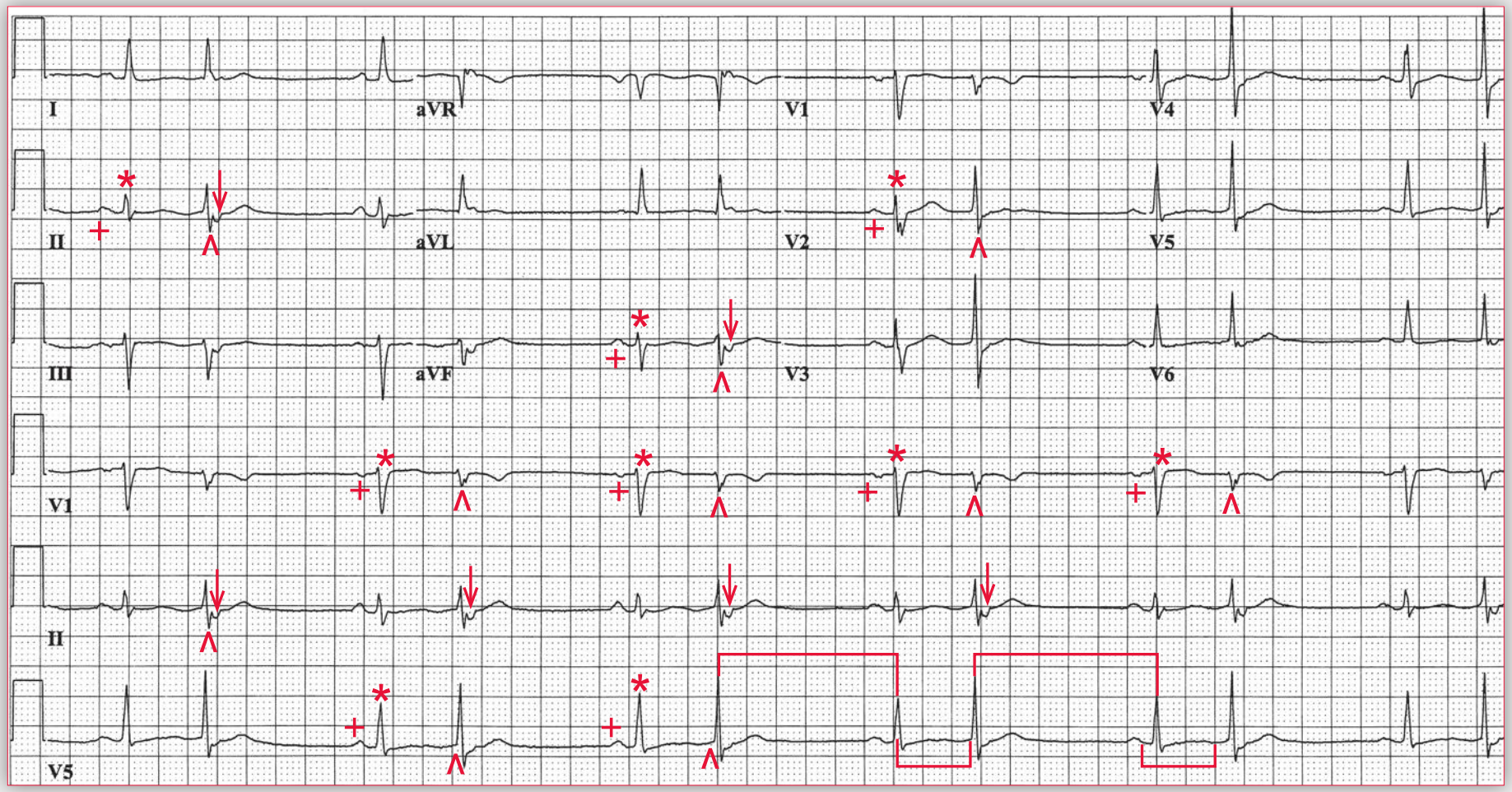
Notes

Practice Case 69

An ECG is obtained from a 47-year-old woman with known familial dilated cardiomyopathy. She has a strong family history of dilated cardiomyopathy with onset in the fifth decade of life. Her ventricular function and left ventricular chamber dimensions are only mildly abnormal. The ECG is obtained as part of a routine exam.

What disturbances of cardiac conduction are noted on the tracing?





ECG 69 Analysis: Normal sinus rhythm, left axis, premature junctional complexes in a bigeminal pattern, retrograde P waves

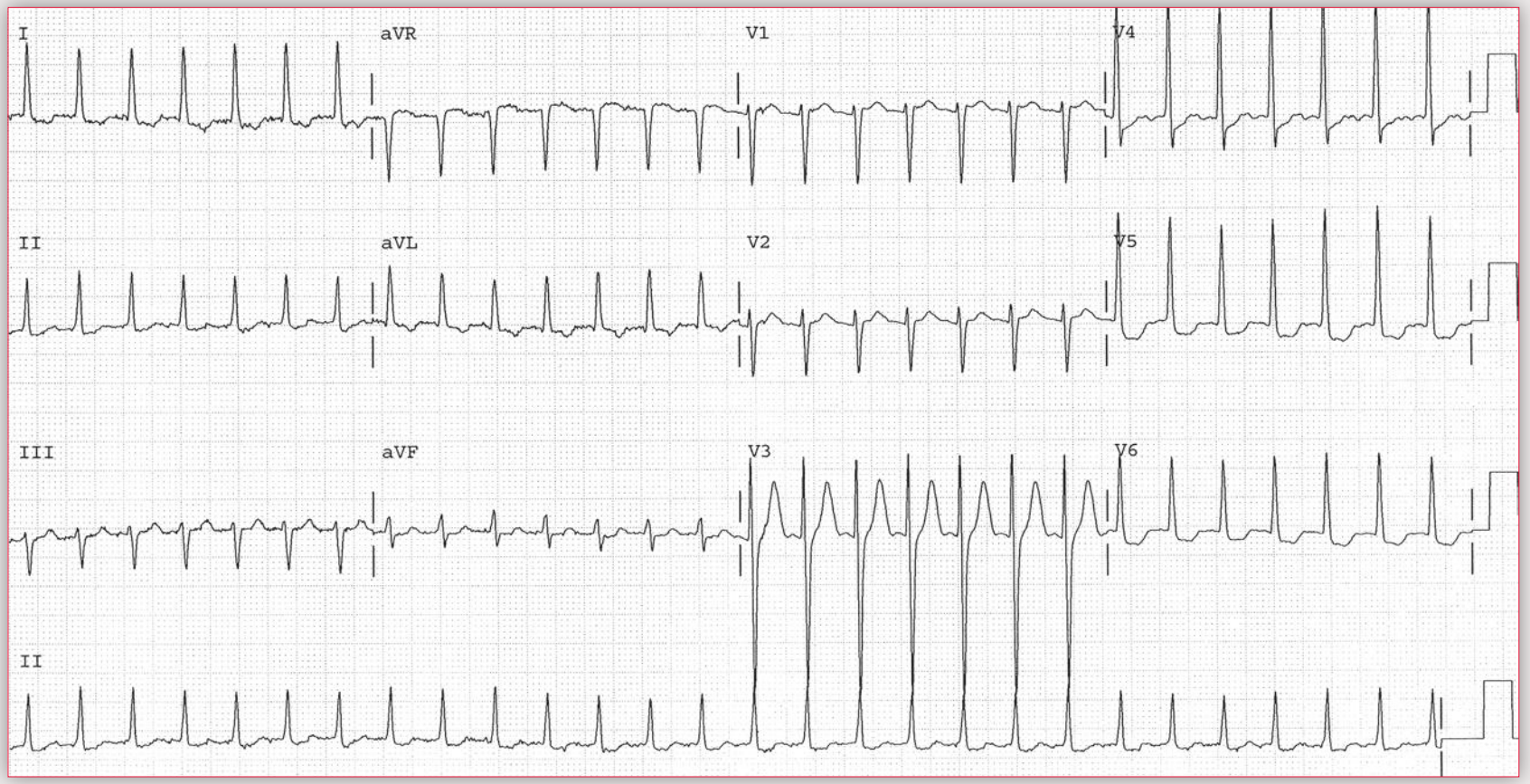
The rhythm is regularly irregular with long and short RR intervals; all the long intervals (⊓) are identical, and all the short intervals (⊔) are identical. Each of the QRS complexes ending the long intervals (*) is preceded by a P wave (+). The P wave is positive in leads I, II, aVF, and V4-V6 and hence is a sinus P wave. There is a stable PR interval (0.16 sec). The QRS complex has a normal duration (0.08 sec) and morphology with a physiologic left axis (positive QRS complex in leads I and II and negative QRS complex in lead aVF). Hence these are normal sinus complexes. The QT/QTc intervals are normal (400/440 msec). After each sinus complex there is an early (premature) QRS complex (^) with a fixed relationship (same RR interval) to the sinus complex (fixed coupling interval [⊔]). These premature complexes have a QRS morphology and duration that are similar to those of the sinus complex, but none is preceded by a P wave. There is, however, a negative P wave (↓) after the premature QRS complex with a short and consistent RP interval. These are, therefore, premature junctional complexes with retrograde P waves. Because every other QRS complex is a premature junctional complex, this is termed junctional bigeminy.

Although the premature junctional complexes have the same QRS morphology and duration as the sinus complexes, they do have a different amplitude in many leads. It is very common for junctional complexes to have a slightly different amplitude and/or axis compared with sinus complexes. This is due to the fact that the junctional complex, which originates from an ectopic focus within the AV junction, penetrates the His-Purkinje system, which is a series of tracts, at a slightly different location compared with the sinus impulse that goes through the AV node. As the location of conduction of the junctional impulse through the His-Purkinje system differs from that of a sinus impulse, the QRS complex originating in the junction may have a slightly different amplitude and/or axis. ■

Practice Case 70

An 82-year-old man presents to his primary doctor with complaints of intermittent palpitations associated with chest discomfort. He has noted episodes lasting seconds to minutes for several weeks now, most often in the late morning after breakfast. The more prolonged episodes are associated with

ECG 70A



Practice Case 70

diaphoresis and chest pressure radiating to his jaw. A review of symptoms is otherwise unremarkable.

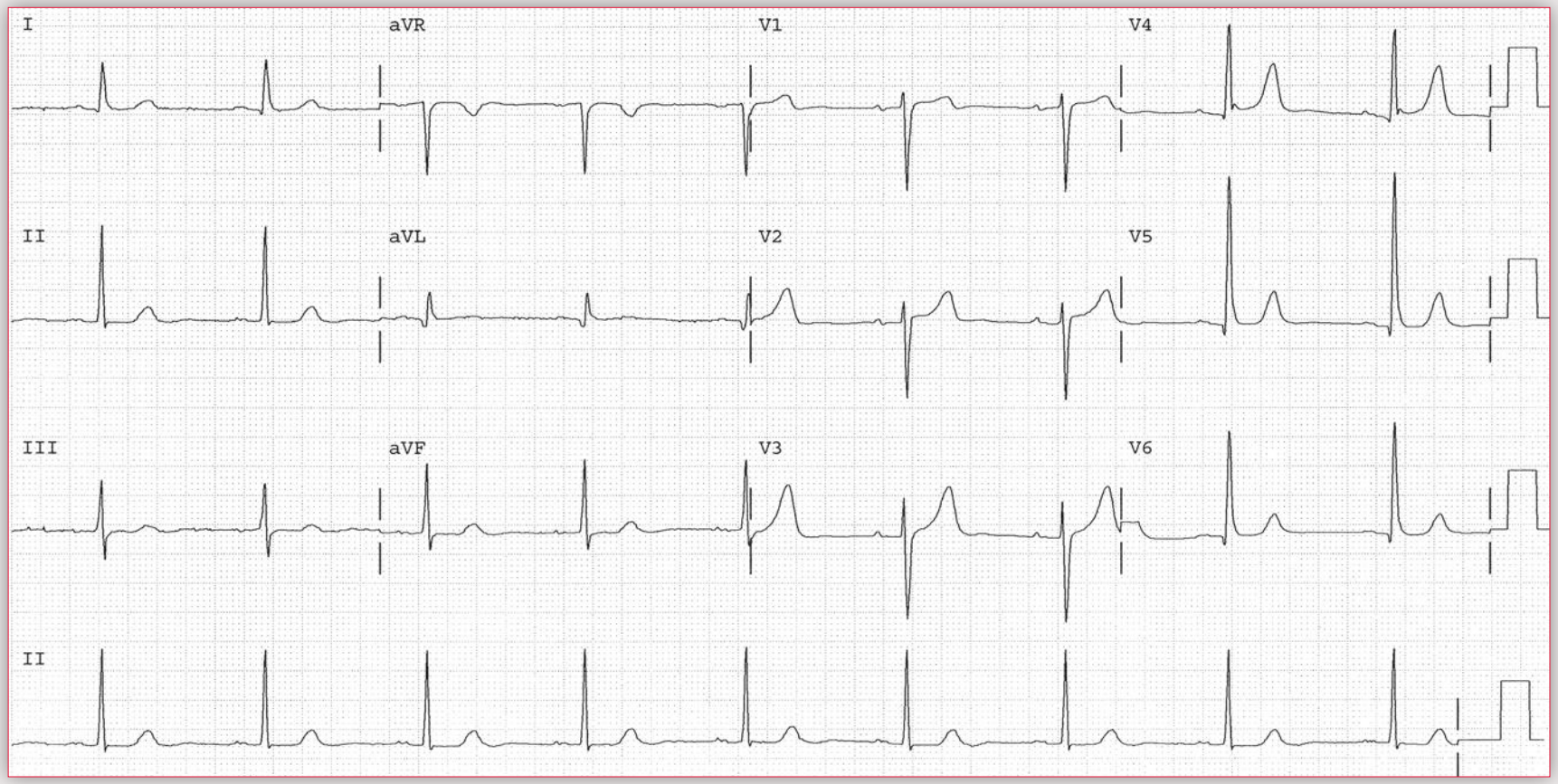
The patient is currently having symptoms. You obtain an ECG (70A). While recording, the patient coughs and you note a change in his heart rate. You obtain a second tracing (ECG 70B).

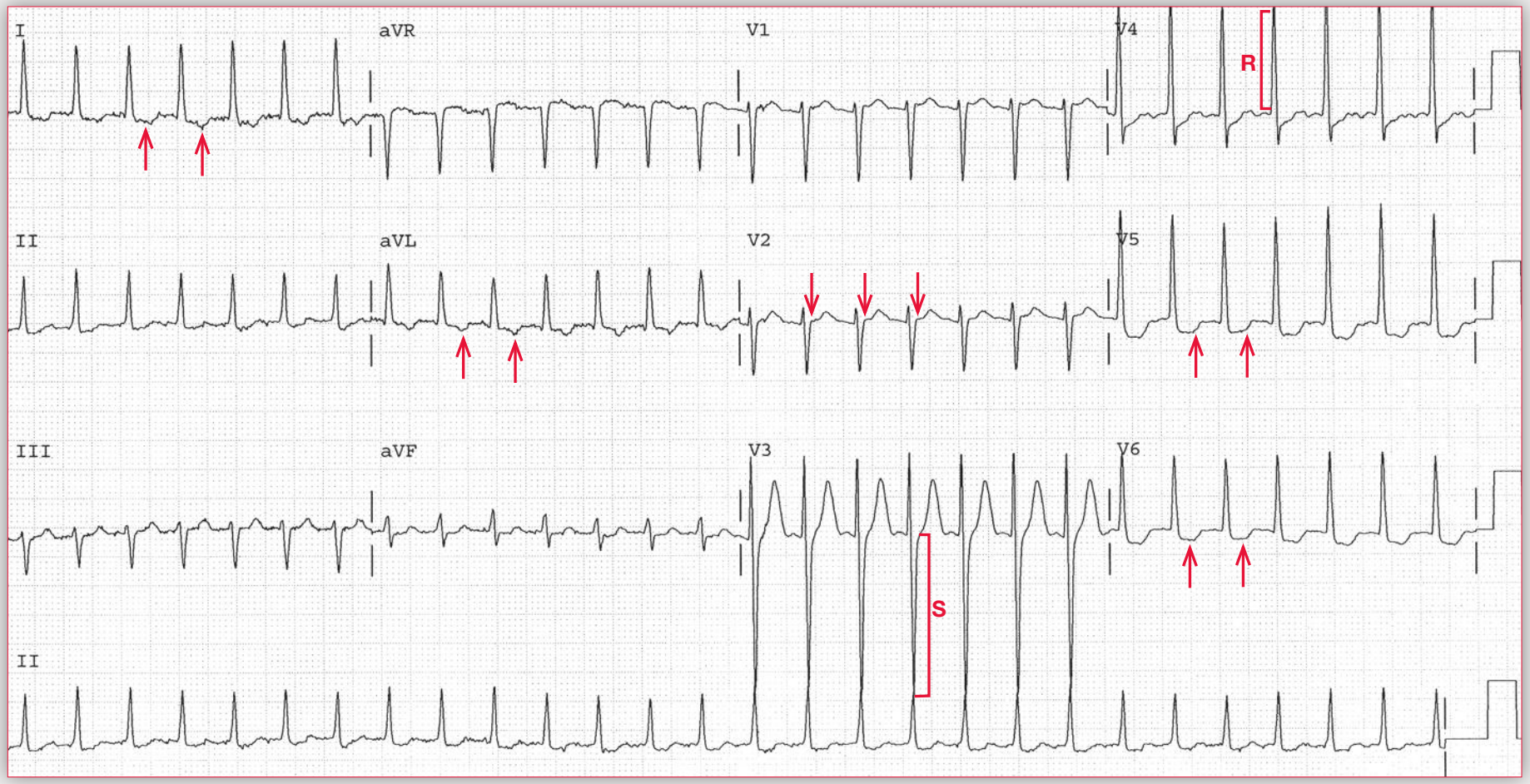
What abnormalities are evident on ECG 70A?

When compared with ECG 70B, what further can be said about the patient's possible ECG diagnosis?

What is the etiology of his chest discomfort?

ECG 70B





ECG 70A Analysis: Narrow complex tachycardia, atrioventricular nodal reentrant tachycardia (no RP tachycardia), left ventricular hypertrophy (LVH), ST-T wave abnormalities

In **ECG 70A**, the rhythm is regular at a rate of 170 bpm. The QRS complex duration is narrow (0.08 sec) and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal, but its amplitude is increased in leads V3 (S-wave depth = 30 mm [ ]]) and V4 (R-wave amplitude = 20 mm [[ ]). This is diagnostic for left ventricular hypertrophy (LVH) (*ie*, S-wave depth in lead V3 + R-wave amplitude in lead V4 = 50 mm based on one of the criteria for LVH [S-wave depth + R-wave amplitude any two precordial leads ≥ 35 mm]). The QT/QTc intervals are normal (260/440 msec). There are ST-T wave abnormalities in leads I, aVL, and V5-V6 (↑) that may be secondary to LVH or the rapid rate.

No P waves are seen before or after any of the QRS complexes. However, there is a very small R′ waveform in leads V1 and V2 (↓) that is at the very end of the QRS complex and is possibly a retrograde P wave. Comparison with an ECG in sinus rhythm would confirm whether this is R′ waveform is due to a right ventricular conduction delay or a retrograde P wave. Nevertheless, it is not clearly a P wave so this is what may be called no RP tachycardia. The most common etiology is AV nodal reentrant tachycardia.

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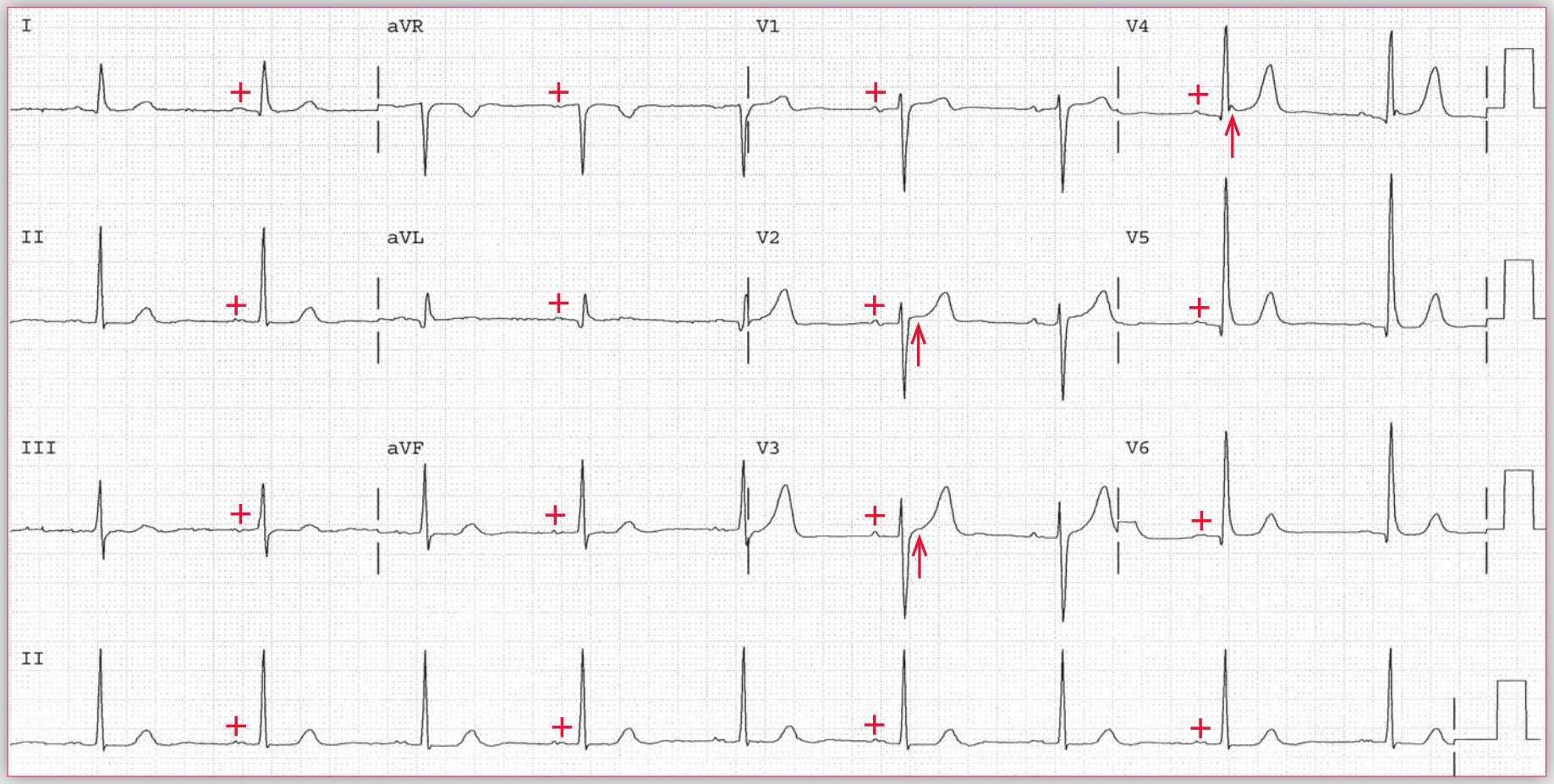


ECG 70B Analysis: Sinus bradycardia, first-degree AV block (prolonged AV conduction), LVH

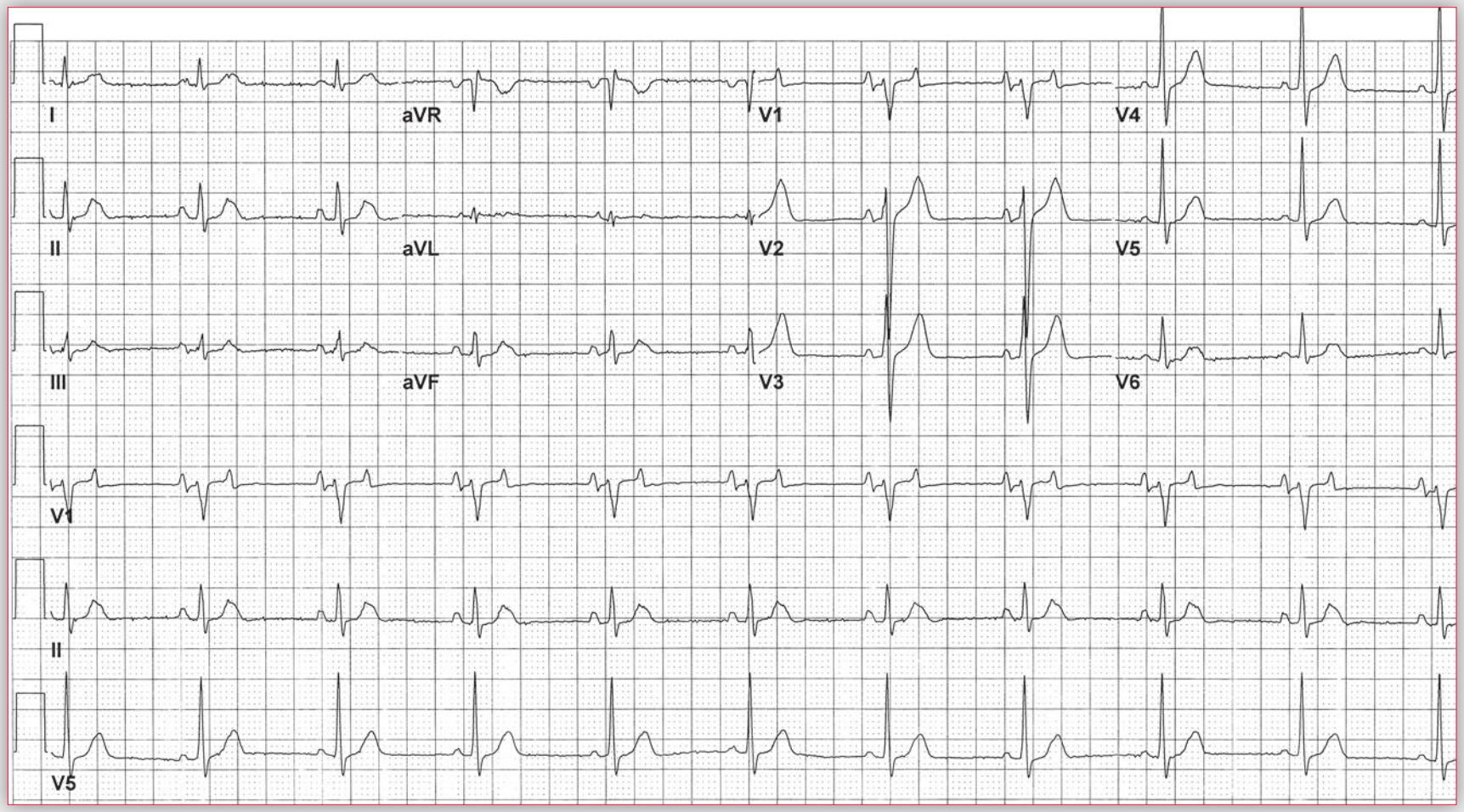
In **ECG 70B** the rate is 54 bpm and the QRS complex morphology, axis, and duration are identical to what is seen in **ECG 70A**. The QT/QTc intervals are normal (440/420 msec). There is a P wave (**+**) before each QRS complex with a stable PR interval (0.20 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence this is sinus bradycardia. The amplitude of the QRS complexes in lead V5 is high (25 mm), consistent with LVH. Associated with the hypertrophy is slight ST-segment elevation in leads V2-V4 (↑), which is termed early repolarization. The R′ waveform superimposed at the end of the QRS complex in leads V1 and V2 in **ECG 70A** is no longer seen, suggesting that the R′ waveform was a retrograde P wave. There are no ST-T wave changes in the apicolateral leads, meaning that the ST-segment changes in **ECG 70A** were likely ischemia, related to the rapid heart rate. Further support for ischemia is the occurrence of chest pressure, characteristic of angina, during the tachycardia. ■

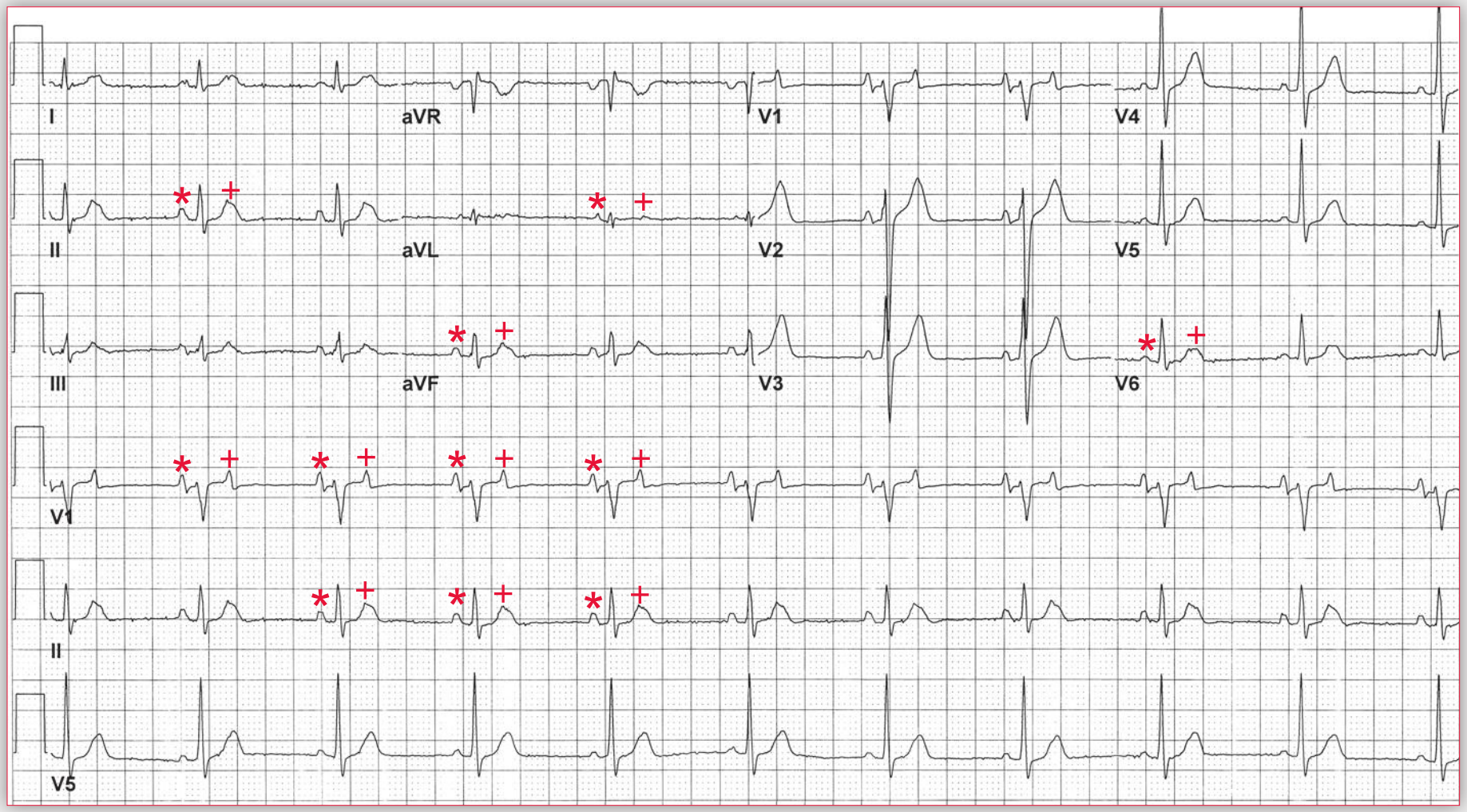
Notes

Practice Case 71

A 78-year-old man is in the neurologic intensive care unit after suffering an intracranial hemorrhage. A cardiology consult is called for a T-wave abnormality.

What does the ECG show?





ECG 71 Analysis: Normal sinus rhythm, atrial bigeminy with blocked premature atrial complexes (blocked premature atrial complexes in a bigeminal pattern)

There is a regular rhythm at a rate of 62 bpm. There is a P wave (*) before each QRS complex, and the PR interval is constant (0.16 sec). The QRS complexes are normal in duration and morphology. After each QRS complex is an early or premature P wave superimposed on the T wave (+), best seen in leads V1, III, and aVF. In other leads the P wave produces a bump on the T wave. The upstroke and downstroke of the T waves should be smooth. Notching, bumps, or irregularities of the T wave suggest superimposed P waves. Hence these are blocked or nonconducted premature atrial complexes occurring in a bigeminal pattern. There is a fixed relationship (0.34 sec) between the sinus P wave and each of the premature atrial waveforms (fixed coupling interval).

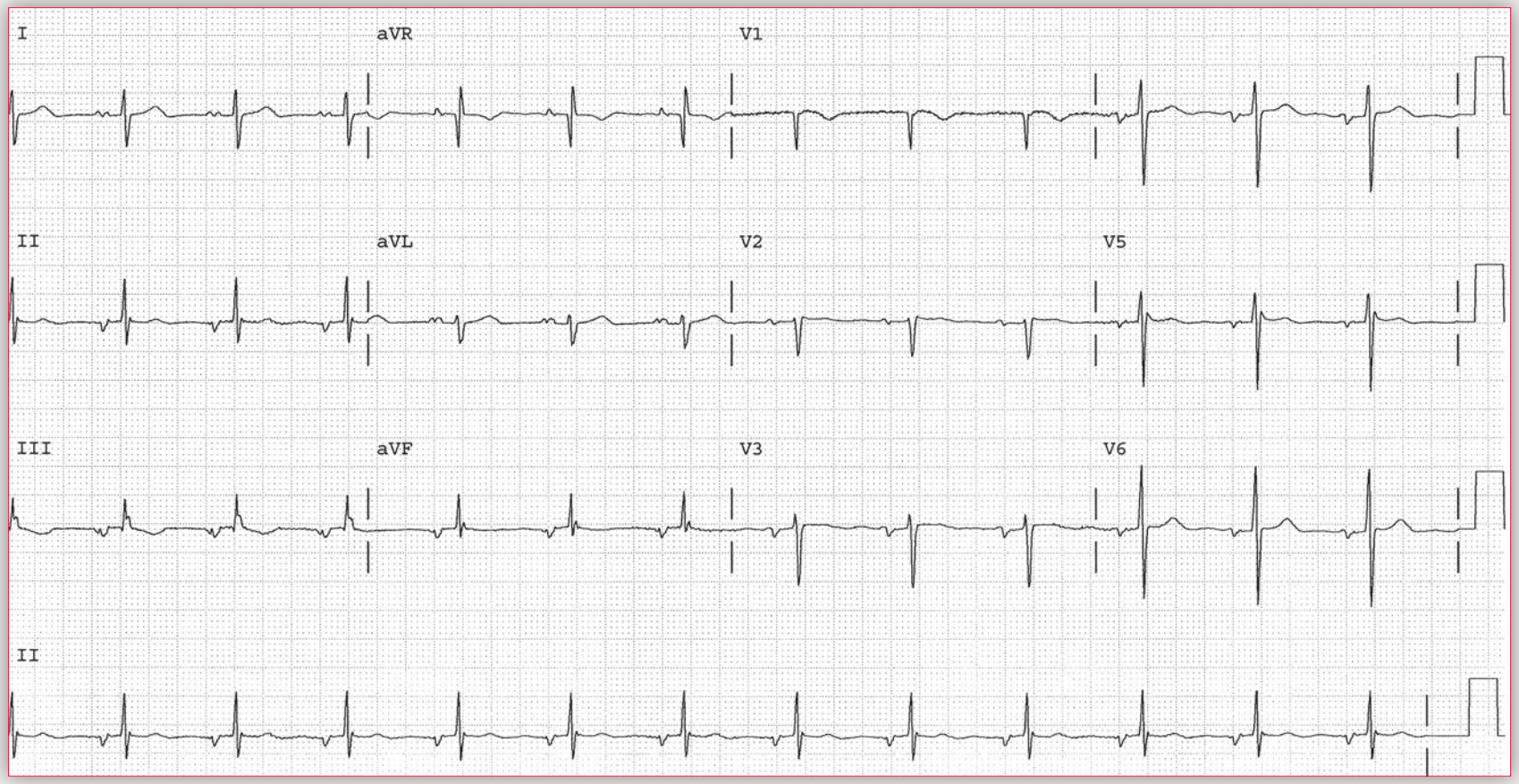
The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/370 msec).

Premature atrial complexes are common and not indicative of heart disease. No therapy is generally required unless symptoms are present or the effective heart rate is slow (and associated with symptoms) as a result of the blocked complexes. ■

Practice Case 72

A 71-year-old woman with ischemic cardiomyopathy (New York Heart Association class III heart failure) presents to her cardiologist for follow-up. She also has mild pulmonary hypertension on the basis of chronic left-sided heart failure. She is taking lisinopril, carvedilol, and digoxin. Her current ECG is shown (ECG 72A). An ECG from a prior visit is also shown (ECG 72B).

ECG 72A



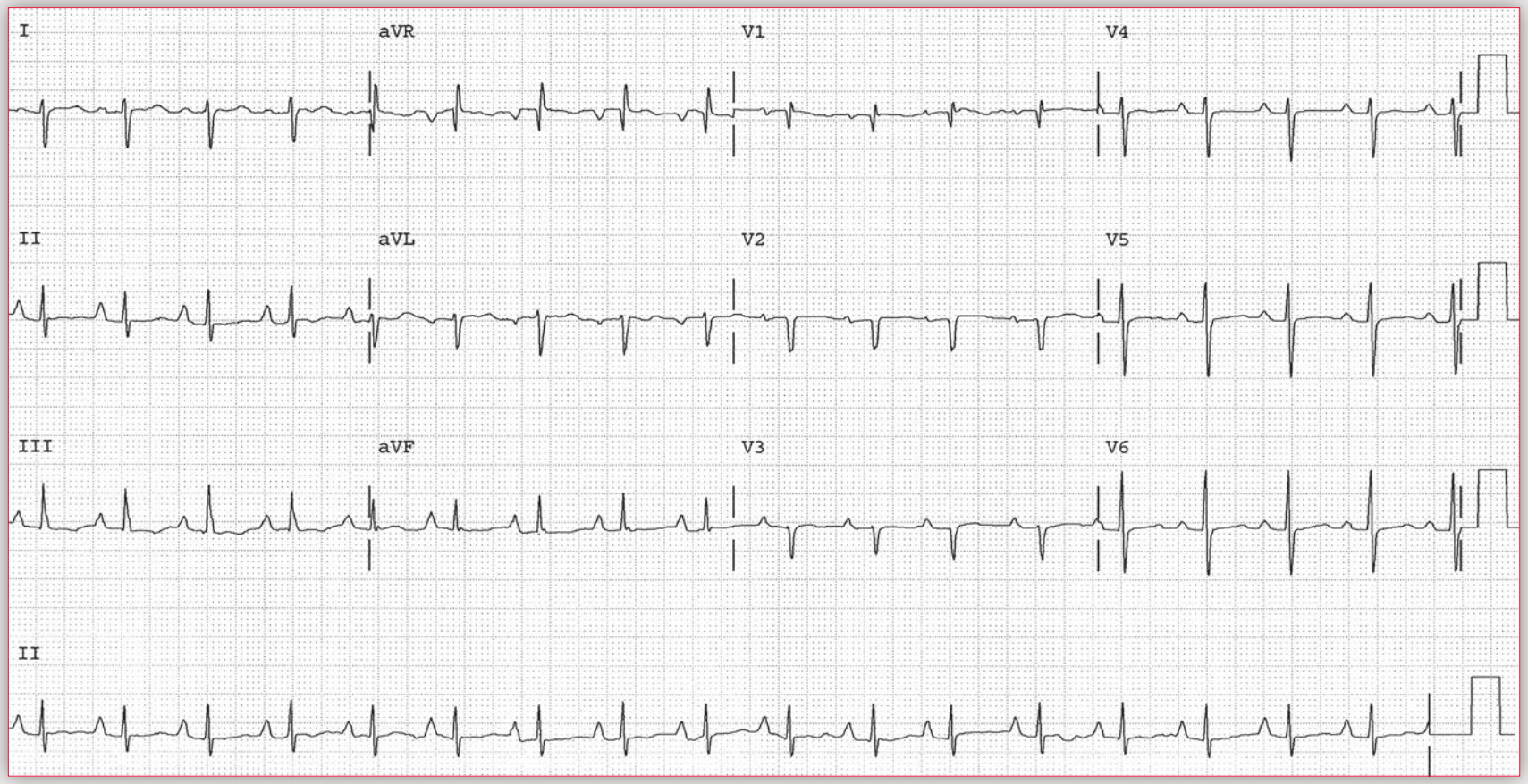
Practice Case 72

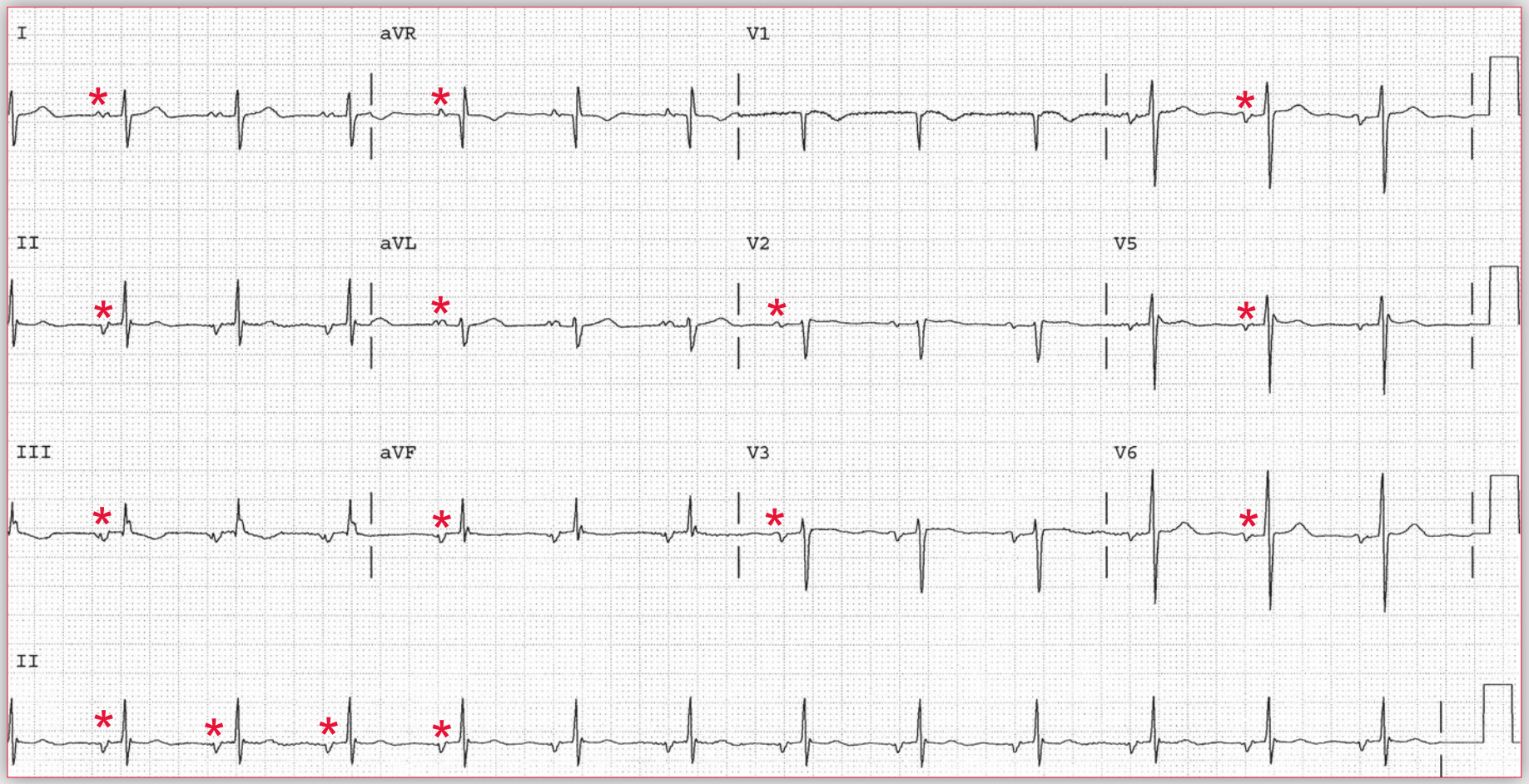
What abnormality is notable on ECG 72A?

Which medication is likely contributing to this abnormality?

What are the major findings on ECG 72B?

ECG 72B



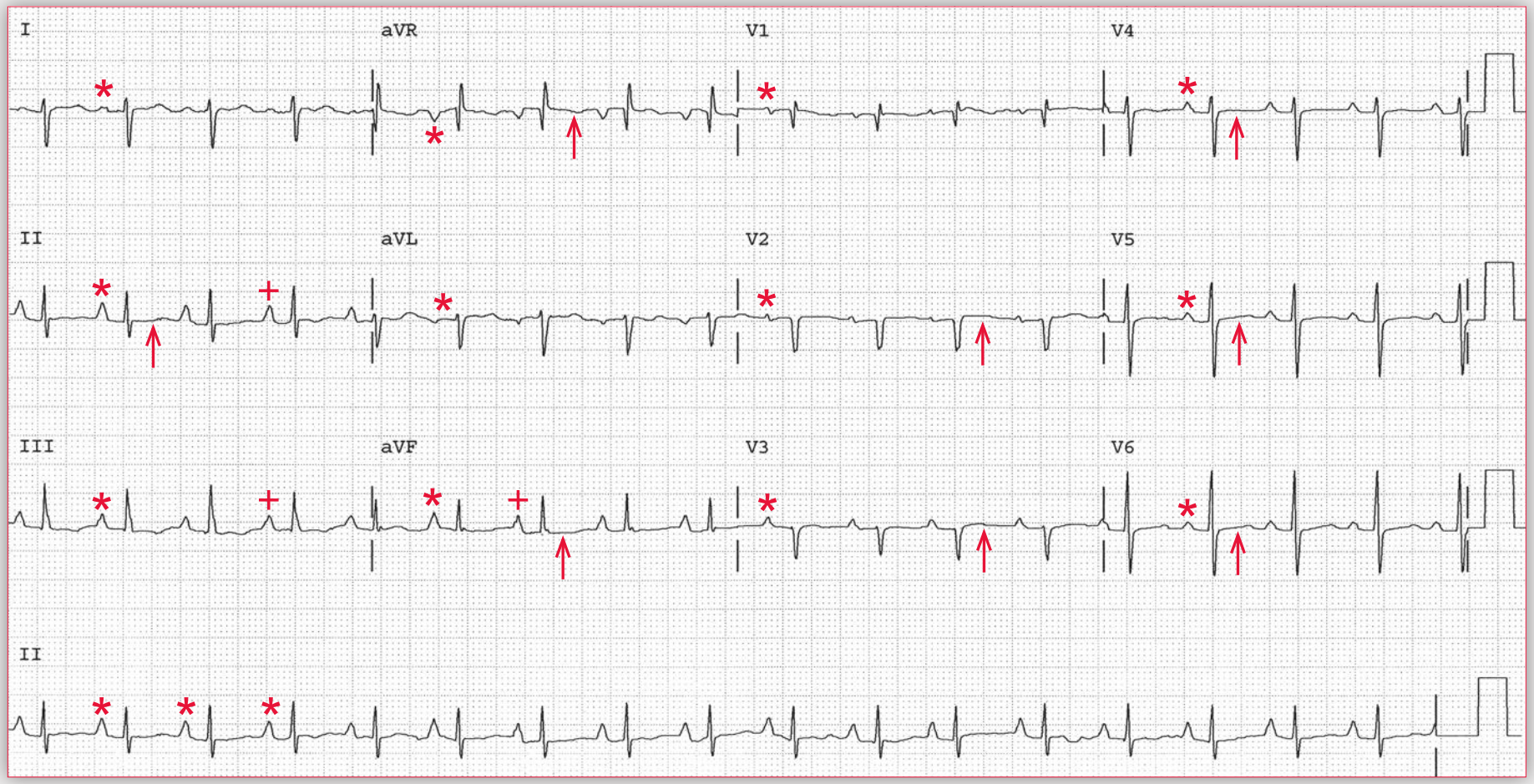


ECG 72A Analysis: Ectopic atrial rhythm, clockwise rotation (poor R-wave progression across the precordium, late transition), left posterior fascicular block

In **ECG 72A** there is a regular rhythm at a rate of 76 bpm. There is a P wave (*) before each QRS complex with a constant PR interval (0.18 sec). The P waves are abnormal and are negative (inverted) in leads II, aVF, and V3-V6. They are not originating in the sinus node but rather in some region of the atrial myocardium. Hence this is an ectopic atrial rhythm.

The QRS complex duration (0.08 sec) and morphology are normal. The axis is slightly rightward, slightly more positive than +90° (slightly negative QRS complex in lead I and positive QRS complex in lead aVF). Etiologies for a right axis include right ventricular hypertrophy (associated with a tall R wave in lead V1 and often a right atrial abnormality or hypertrophy), lateral wall myocardial infarction (which has a Q wave in leads I and aVL, Wolff-Parkinson-White pattern associated with a short PR interval and a delta wave), right–left arm lead switch (associated with a negative P wave and T wave in leads I and aVL), dextrocardia (which has a pattern of right–left arm lead switch as well as reverse R-wave progression across the precordium), and left posterior fascicular block (which is diagnosed when there is no other etiology for the right axis). As none of the etiologies for a right axis is present, this is a left posterior fascicular block. There is poor R-wave progression in leads V1-V2 and late transition (R/S = 1) in lead V6. These features, which represent the electrical axis in the horizontal plane, are the result of clockwise rotation, which is determined by imagining the heart as viewed from under the diaphragm. When the electrical axis is rotated in a clockwise direction, the left ventricular forces are shifted posteriorly and seen in the more lateral precordial leads, accounting for poor R-wave progression and late transition. The QT/QTc intervals are normal (360/410 msec).

Ectopic atrial rhythms are uncommon. They may result from increased sympathetic tone that enhances an ectopic focus. They may also result from depression of sinus node activity with the occurrence of an escape atrial focus. These conditions may be the result of digoxin, which can slow sinus node activity as a result of its peripheral vagal effect and also increase the automaticity of ectopic atrial foci as a result of an increase in central sympathetic neural outputs that occur in the setting of digoxin excess. Uncommonly, an atrial rhythm may be the result of a reentrant circuit within the atrial myocardium.



ECG 72B Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), left posterior fascicular block, clockwise rotation (poor R-wave progression across the precordium), right atrial hypertrophy (or abnormality)

ECG 72B shows a regular rhythm at a rate of 110 bpm. There is a P wave (*) before each QRS complex with a constant PR interval (0.24 sec). The P wave is positive (upright) in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia with first-degree AV block. The P waves are tall and peaked in leads II, III, and aVF (+), suggesting the presence of right atrial hypertrophy or a right atrial abnormality. The axis in the frontal plane is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). As indicated, a right axis may be due to right ventricular hypertrophy, right–left arm lead switch, Wolff-Parkinson-White pattern, a lateral wall myocardial infarction, or dextrocardia. In the absence of any of these abnormalities, the right axis is termed left posterior fascicular block.

The P wave is positive in leads I and aVL, so the right axis is not due to lead switch or dextrocardia as these conditions would be associated with a negative P wave in these leads. The PR interval is normal and the QRS complex does not have a delta wave, eliminating Wolff-Parkinson-White pattern. There are no Q waves in leads I or aVL, excluding a lateral wall myocardial infarction. However, it is unclear whether the right axis is due to right ventricular hypertrophy or a left posterior fascicular block. Although there is no evidence of right ventricular hypertrophy (*ie*, a tall R wave in lead V1), right atrial hypertrophy and a right axis are associated abnormalities that suggest right ventricular hypertrophy. A diagnosis of left posterior fascicular block is one of exclusion; that is, there is no other cause for the right axis.

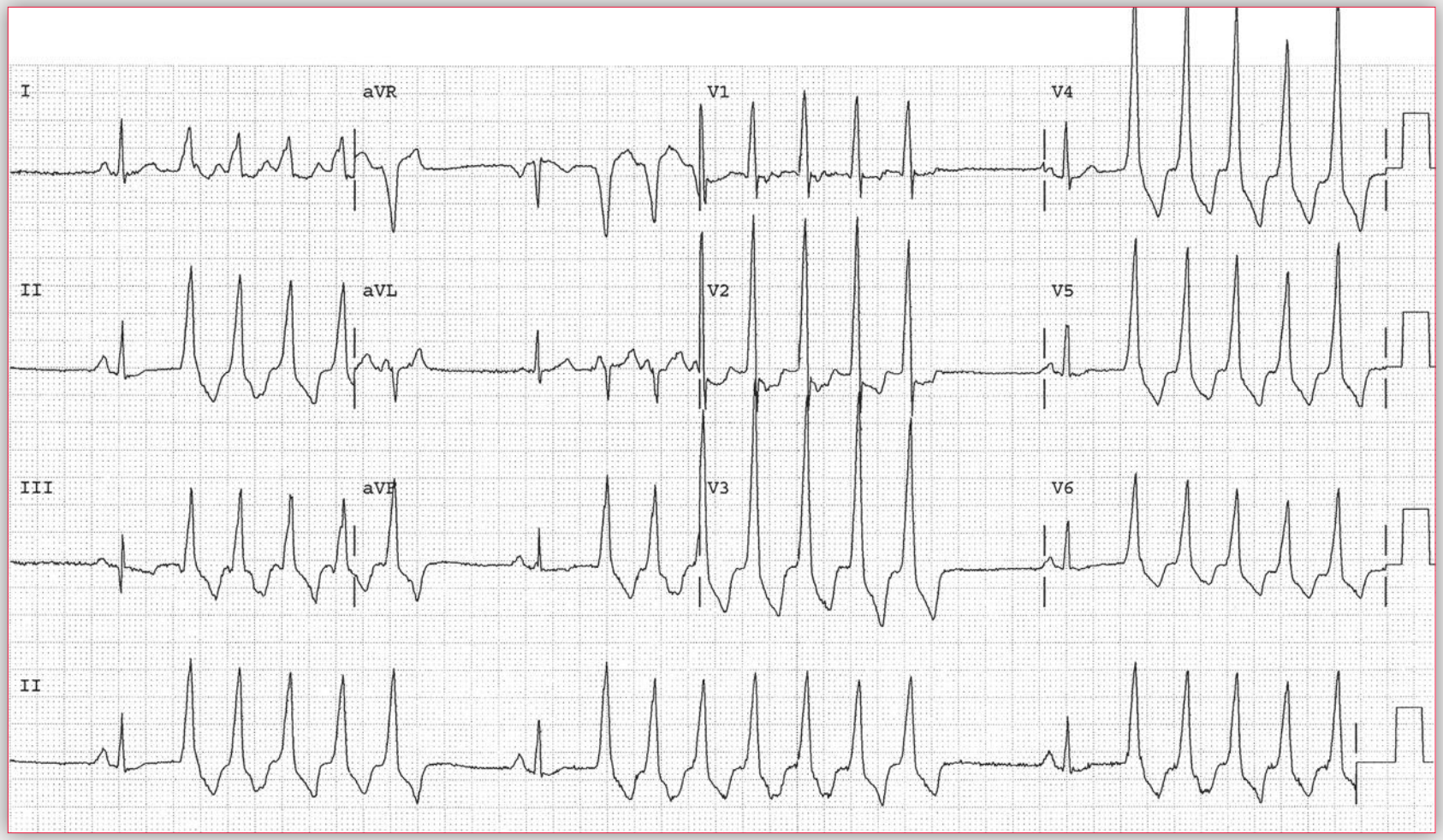
The QRS complex duration and QT/QTc intervals are the same as in **ECG 72A**. However, on this ECG it appears that there are no R waves in leads V1-V3 but rather there are Q waves in leads V1-V2, consistent with an anteroseptal myocardial infarction. However, this pattern may also be seen in normal subjects, especially women, as a result of attenuation of anterior forces by breast tissue. Also present is nonspecific T-wave flattening (↑). ■

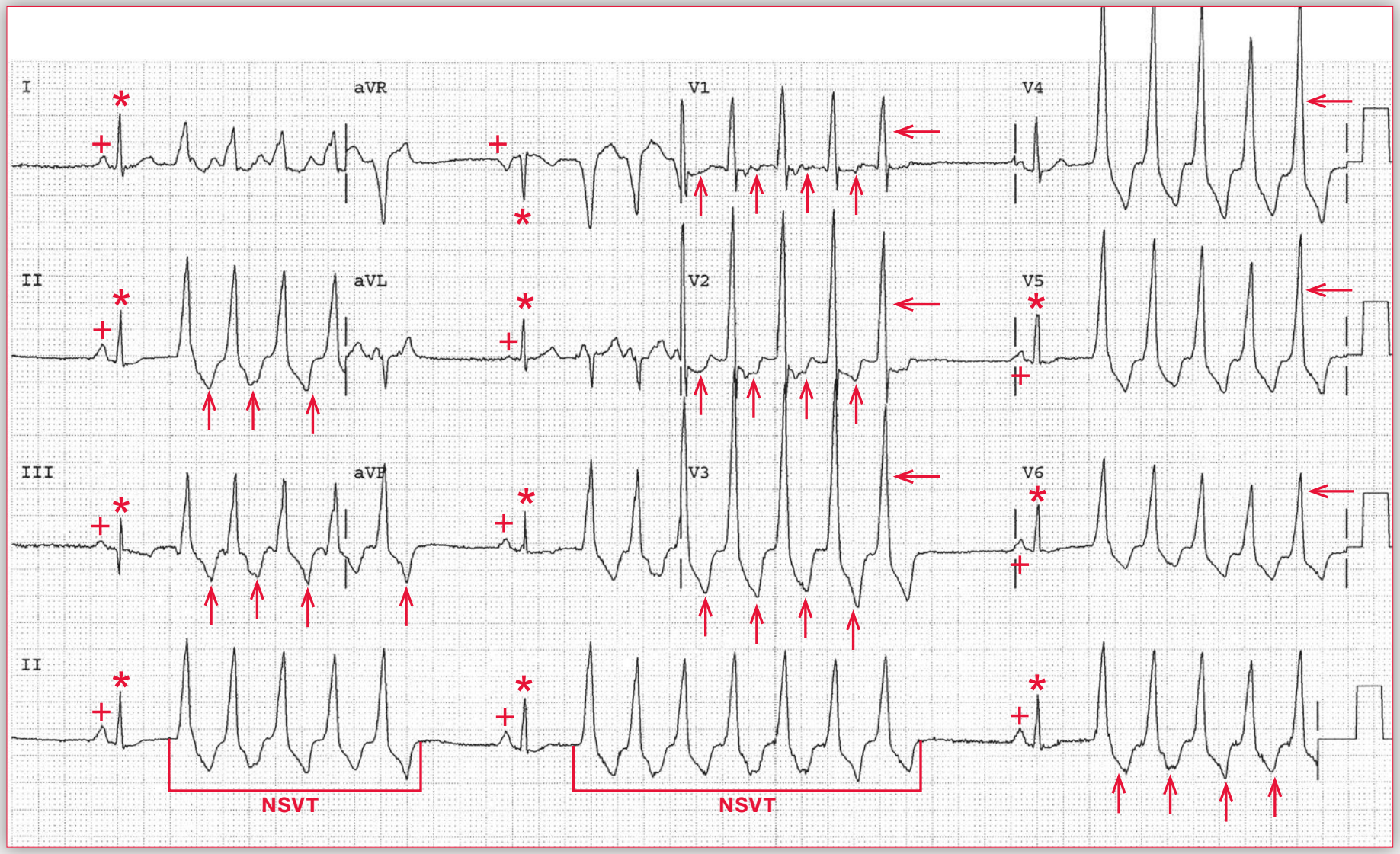
Notes

Practice Case 73

Two medical residents are debating whether the following ECG, which shows wide complex tachycardia, signifies ventricular tachycardia or aberrantly conducted supraventricular tachycardia.

What are the important findings that make the diagnosis?





ECG 73 Analysis: Sinus rhythm, nonsustained ventricular tachycardia

Three narrow QRS complexes (0.08 sec) can be seen (*). Each one is preceded by a P wave (+) and has a constant PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). Following the sinus complexes are runs of five to seven wide QRS complexes (0.16 sec) at a rate of 150 bpm (⊔). All of these complexes have a similar QRS morphology; the morphology is abnormal and not typical for either a right or left bundle branch block. There are no P waves associated with any of the wide QRS complexes. However, there are subtle differences in the ST-T wave morphology, especially well seen in leads II, III, and V1-V2 (↑). These may represent differences in repolarization or possibly superimposed P waves.

Regardless of the etiology, these changes are seen with ventricular tachycardia and not with supraventricular tachycardia with aberration. With supraventricular tachycardia, regardless of etiology (sinus, atrial, or AV nodal–junctional), each impulse is conducted to the ventricle via the same pathway, which may be the normal AV node–His-Purkinje system or an accessory pathway. Hence every QRS complex and every ST segment and T wave are identical. Since ventricular tachycardia results from an abnormal circuit within the ventricular myocardium, the activation of the ventricle bypasses the normal Purkinje system and is via an abnormal pathway. Hence the activation sequence may be variable, accounting for the subtle differences in QRS complex morphology and ST segments and T waves.

In addition, there is positive QRS complex concordance across the precordium (tall R wave in leads V1-V6) (←), which is also seen with QRS complexes of ventricular origin. Positive concordance is seen when there is direct ventricular myocardial activation (*ie*, ventricular complex, paced complex, or ventricular activation via an accessory pathway). No type of conduction through the normal His-Purkinje system is associated with this pattern. It should be noted that negative QRS complex concordance is not helpful as this could be a left bundle branch block.

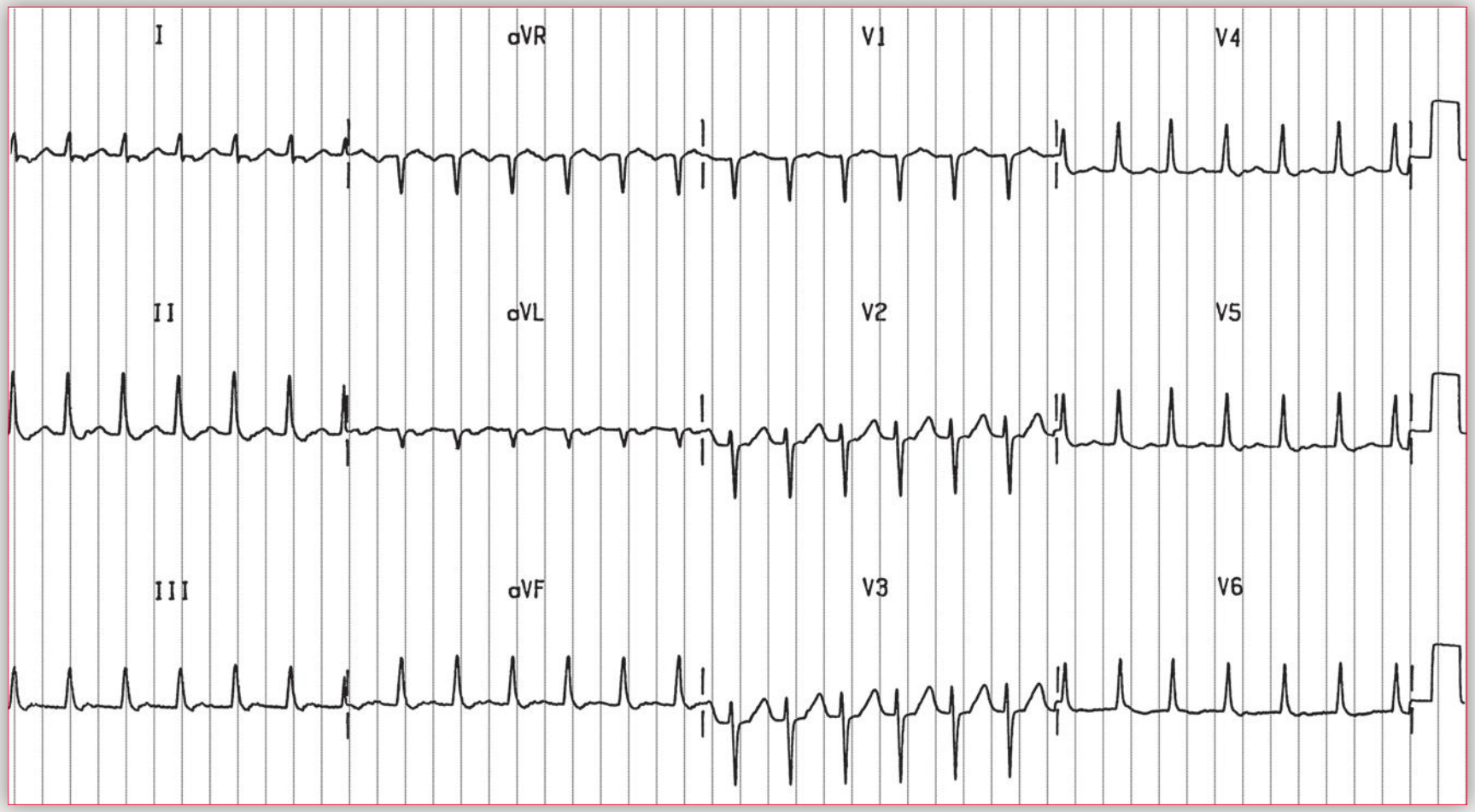
Hence this is monomorphic nonsustained ventricular tachycardia, defined as a ventricular rhythm at a rate of 100 bpm or higher with three or more complexes lasting up to 30 seconds.

Therefore, findings that establish ventricular tachycardia as the etiology are the presence of changes in ST-T wave morphology, possibly AV dissociation, and positive QRS complex concordance across the precordium. ■

Practice Case 74

A 26-year-old medical resident develops palpitations during morning rounds. She takes a moment to sit down and relax, but her heart continues to race. She has a sensation of pounding in her neck and feels

ECG 74A



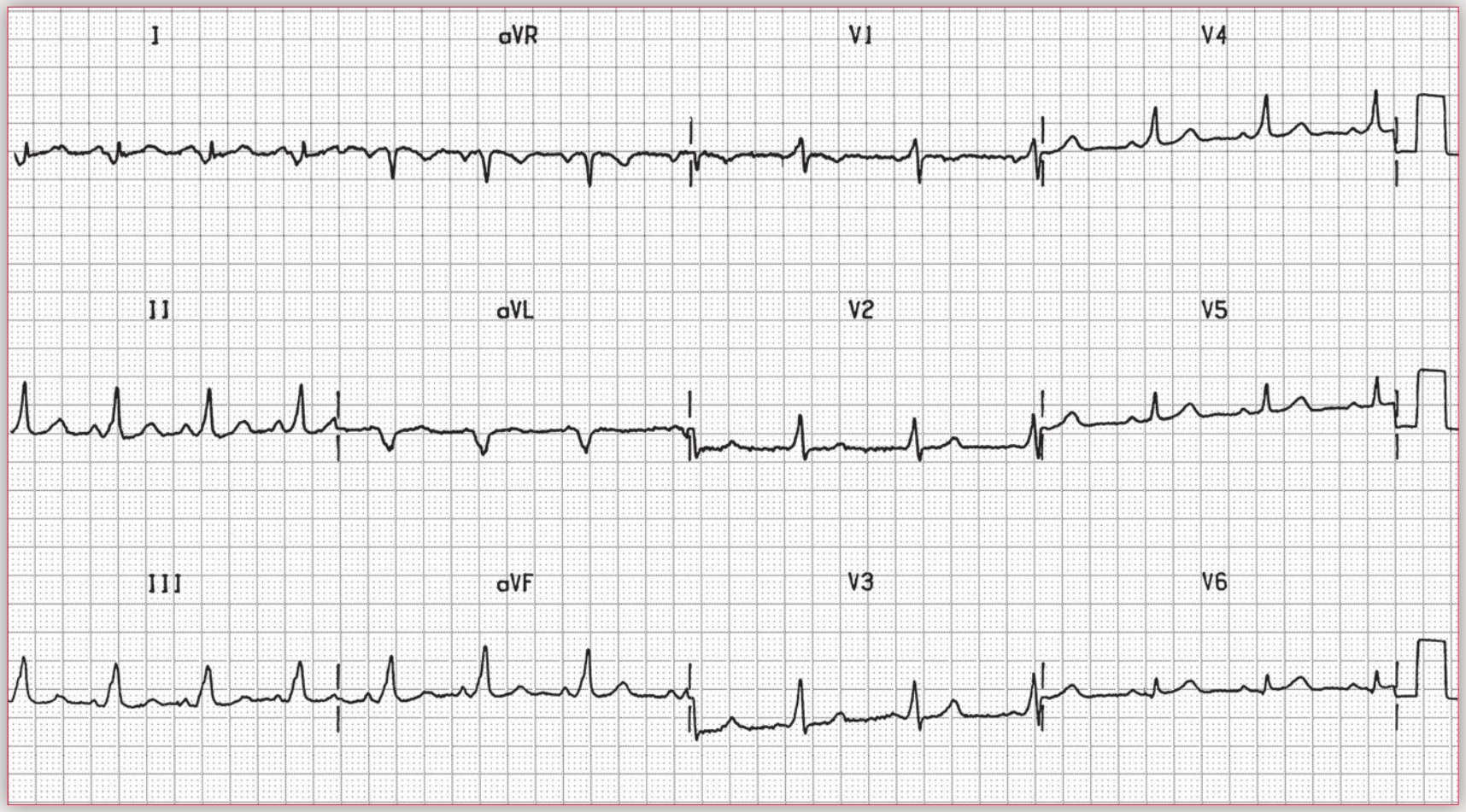
Practice Case 74

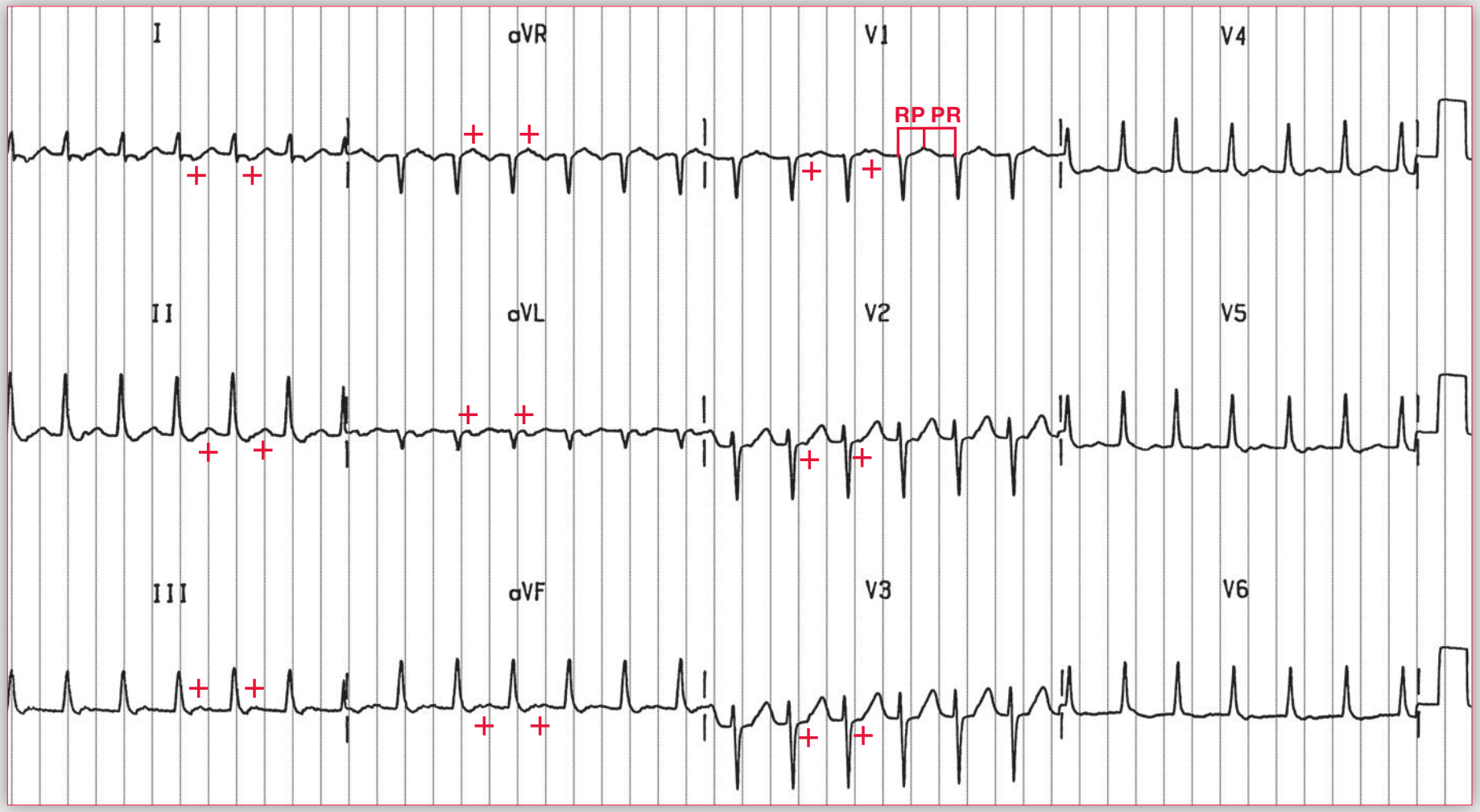
anxious and mildly dyspneic. She is brought to the emergency department by one of her senior residents, who obtains an ECG (74A). After appropriate therapy her heart rate slows and another ECG is obtained (74B).

What is the likely diagnosis?

What can be done clinically to aid in the diagnosis?

ECG 74B





ECG 74A Analysis: Short RP tachycardia, narrow QRS complex tachycardia, orthodromic atrioventricular reentrant tachycardia

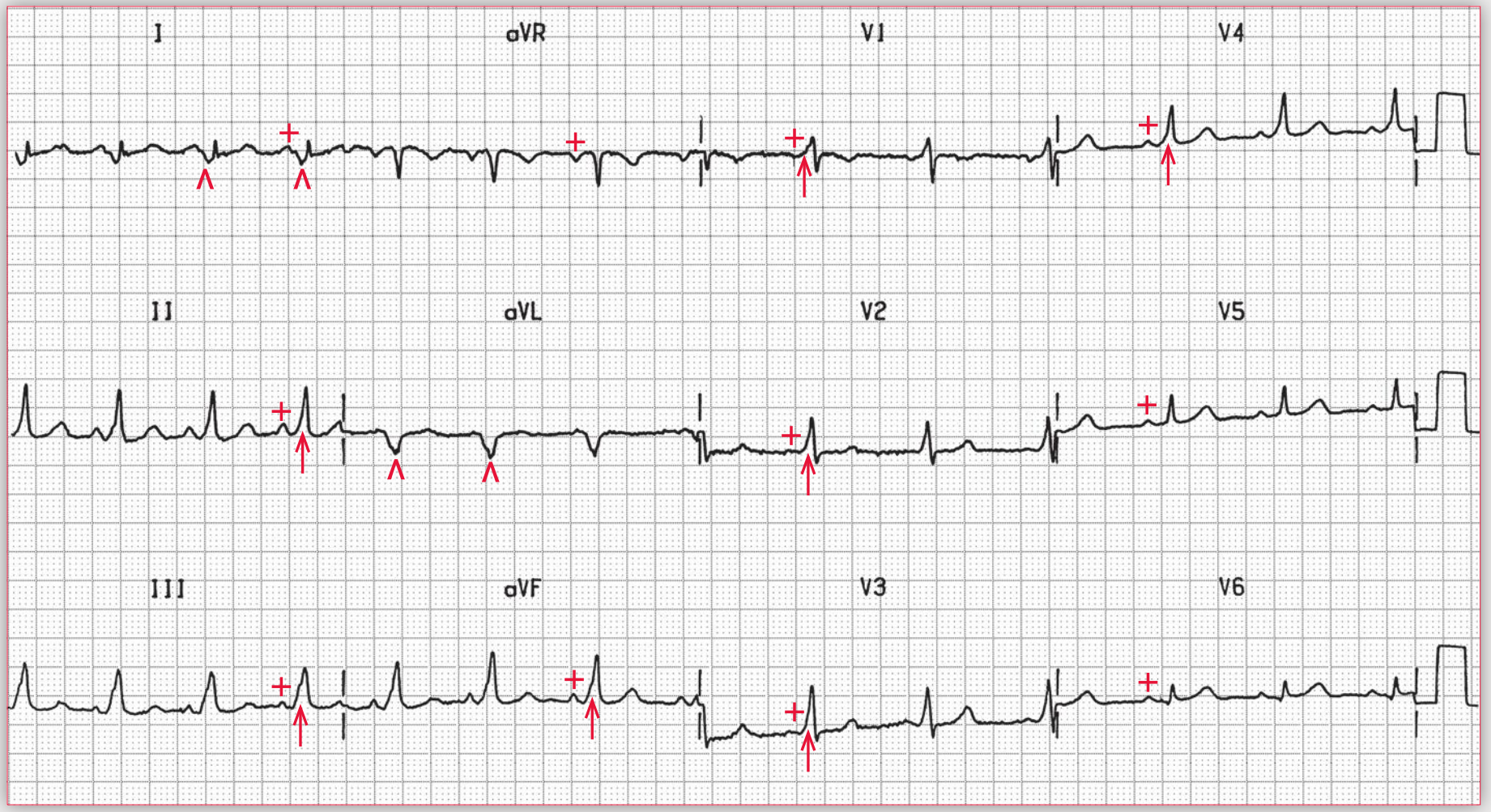
ECG 74A shows a regular rhythm at a rate of 150 bpm. The QRS complex duration is normal (0.08 sec) and there is a normal axis in the frontal plane, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (290/460 msec). No obvious P waves are seen before or after any QRS complex. However, there are notches in the ST segment (+), seen best in leads I, III, aVR, aVL, aVF, and V1-V3. The ST segment should be smooth; notches or bumps within the ST segment suggest a superimposed P wave. The notches appear to be negative in leads I and aVF. These are negative P waves and hence this is short RP tachycardia with retrograde atrial activity (diagnosed by the negative P wave in lead aVF). There are a number of potential etiologies for this, including:

- Sinus tachycardia with a long PR interval
- Atrial flutter with 2:1 AV block
- Ectopic junctional tachycardia
- Atrial tachycardia with a long RP interval
- Orthodromic atrioventricular reentrant tachycardia (AVRT)
- Typical atrioventricular nodal reentrant tachycardia (AVNRT) (slow-fast), in which the retrograde limb (fast pathway) of the circuit conducts relatively slowly (thus making the retrograde P wave visible after the QRS complex). This is uncommon and has often been called slow-slow AVNRT.

It is not possible to establish the etiology of the arrhythmia based on this one ECG. However, sinus tachycardia is not the etiology as the P wave is negative in lead aVF, meaning that there is either a low atrial focus or retrograde atrial activation. Intravenous administration of adenosine or the use of a vagal maneuver can help establish the diagnosis. These interventions affect sinus node automaticity and/or AV nodal conduction properties. In the case of sinus tachycardia, there would be a gradual decrease and then an increase in rate as a result of a vagal maneuver. In the case of atrial tachycardia or atrial flutter there would be no change in atrial rate, but there could be the development of transient AV block and hence a slowing of ventricular rate as a result of either enhanced vagal tone or adenosine. With AV block atrial activity would be exposed and the rate and morphology of the atrial waveforms could be established. An atrial rate over 260 bpm would be diagnostic for atrial flutter. If the rate were less than 220 bpm the rhythm might be either atrial tachycardia or atrial flutter. The morphology of the atrial waveforms is useful in establishing the etiology. In atrial tachycardia there is a distinct P wave with an isoelectric baseline between each P wave as this is generally due to an ectopic focus. In atrial flutter, which is due to a reentrant mechanism, there is continuous electrical activity creating a constant undulation of the atrial waveforms without an isoelectric period between each waveform.

If the rhythm were an ectopic junctional tachycardia, AVNRT, or AVRT, there would be no change or termination of the arrhythmia. If after termination of the arrhythmia the QRS complex were associated with a short PR interval and widened QRS complex, the diagnosis would be Wolff-Parkinson-White pattern and hence the arrhythmia would be AVRT. A short PR interval and normal QRS complex would establish AVRT due to Lown-Ganong-Levine pattern, while a normal PR interval

continues



ECG 74B Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern

and normal QRS complex would establish AVNRT or a concealed bypass tract as the etiology.

Also of use would be the mode of termination of the arrhythmia. Atrial arrhythmias (*ie*, atrial tachycardia or atrial flutter) terminate with the absence of atrial activity as the atrial focus or atrial mechanism ceases its activity and the arrhythmia terminates. In contrast, junctional tachycardia, AVNRT, and AVRT terminate with a nonconducted P wave (assuming there is evidence of atrial activity on the ECG). With these arrhythmias, the ectopic focus or reentrant circuit fails to produce an impulse but there has already been retrograde atrial activation from the previous impulse.

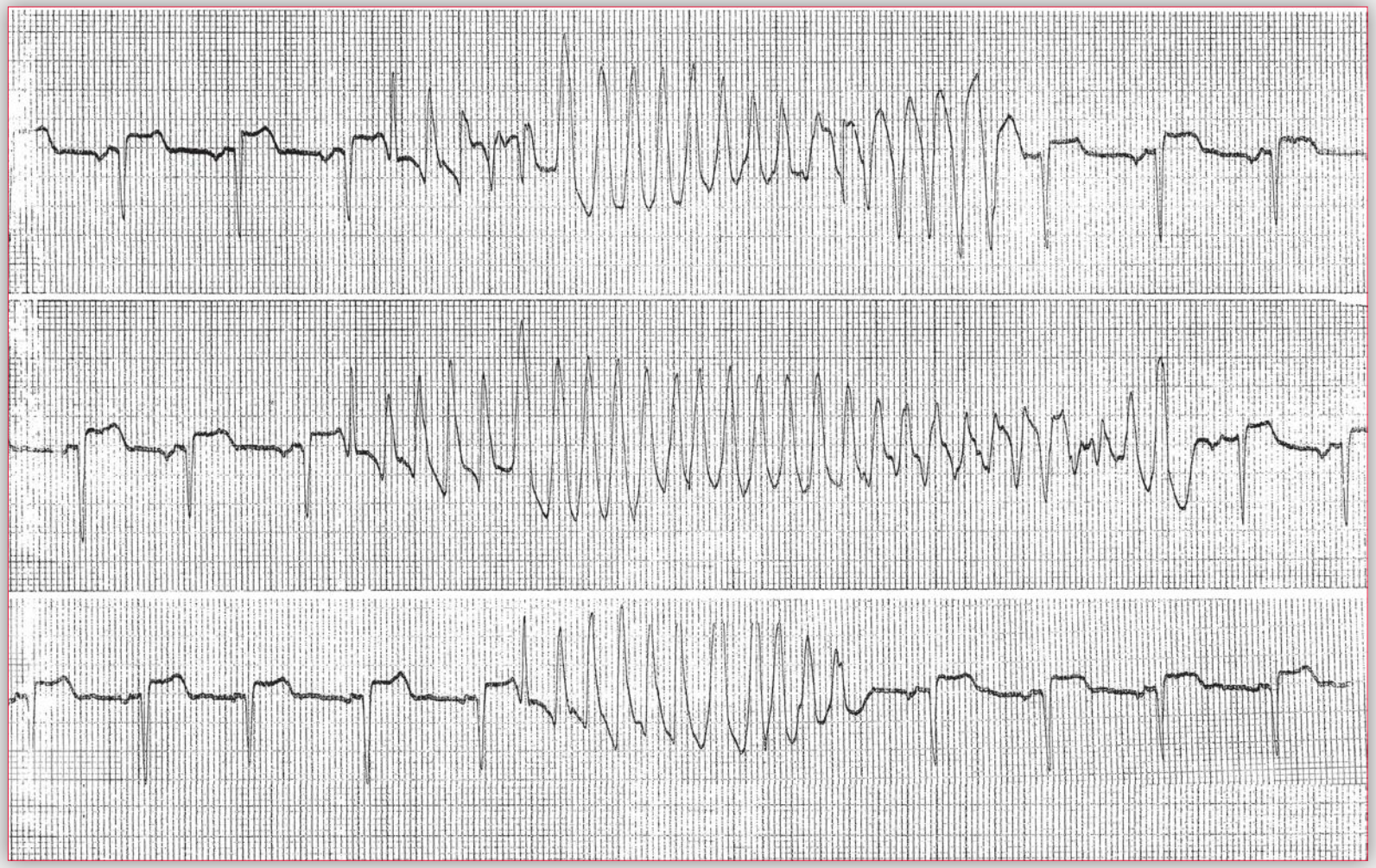
In **ECG 74B** there is a regular rhythm at a rate of 94 bpm. There is a P wave (**+**) before each QRS complex with a constant, but short, PR interval (0.12 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm. The QRS complex duration, as measured at the base of the QRS complex, is increased (0.16 sec). This is due to a wide and slurred upstroke of the QRS complex (↑), particularly evident in leads II, III, aVF, and V1-V4. This is called a delta wave and the pattern seen is typical for Wolff-Parkinson-White. The bypass tract is left lateral as there is a positive delta wave in lead V1 and a pseudo lateral wall infarction pattern, with Q waves in leads I and aVL (^). Hence the arrhythmia in **ECG 74A** is an orthodromic AVRT; that is, activation of the ventricle is via the normal AV node–His Purkinje system (hence the narrow and normal QRS complex), and the retrograde impulse conduction back to the atrium (resulting in retrograde atrial activation) is via the accessory pathway. The QT/QTc intervals are prolonged (420/520 msec) but are normal when corrected for the prolonged QRS complex duration (360/440 msec). ■

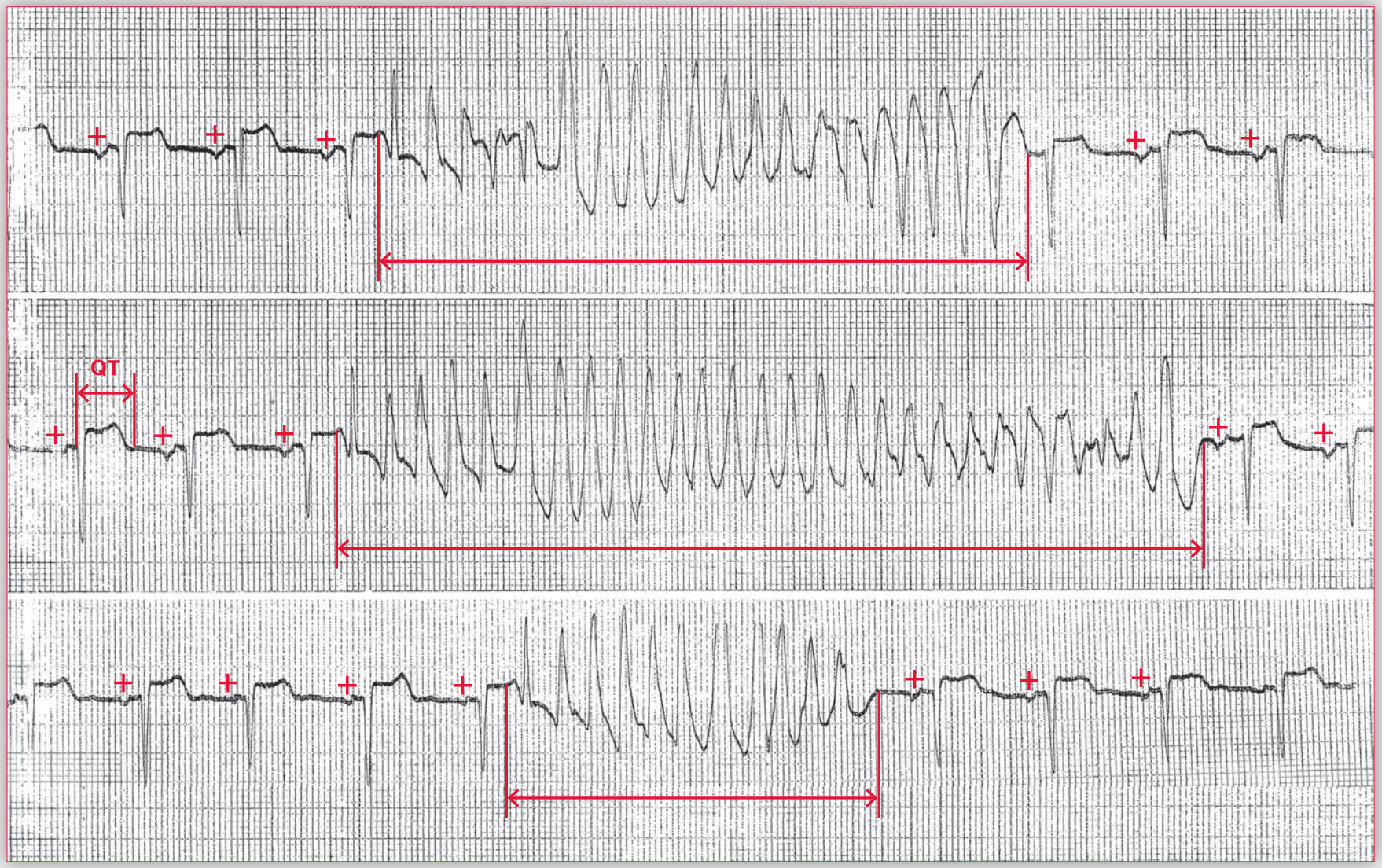
Notes

Practice Case 75

A 72-year-old woman presents to the emergency department following 24 hours of chest pain and dyspnea. She also complains of intermittent near-syncope. The following rhythm strips are obtained.

What is the arrhythmia?





ECG 75 Analysis: Nonsustained polymorphic ventricular tachycardia with a normal QT interval, normal sinus rhythm

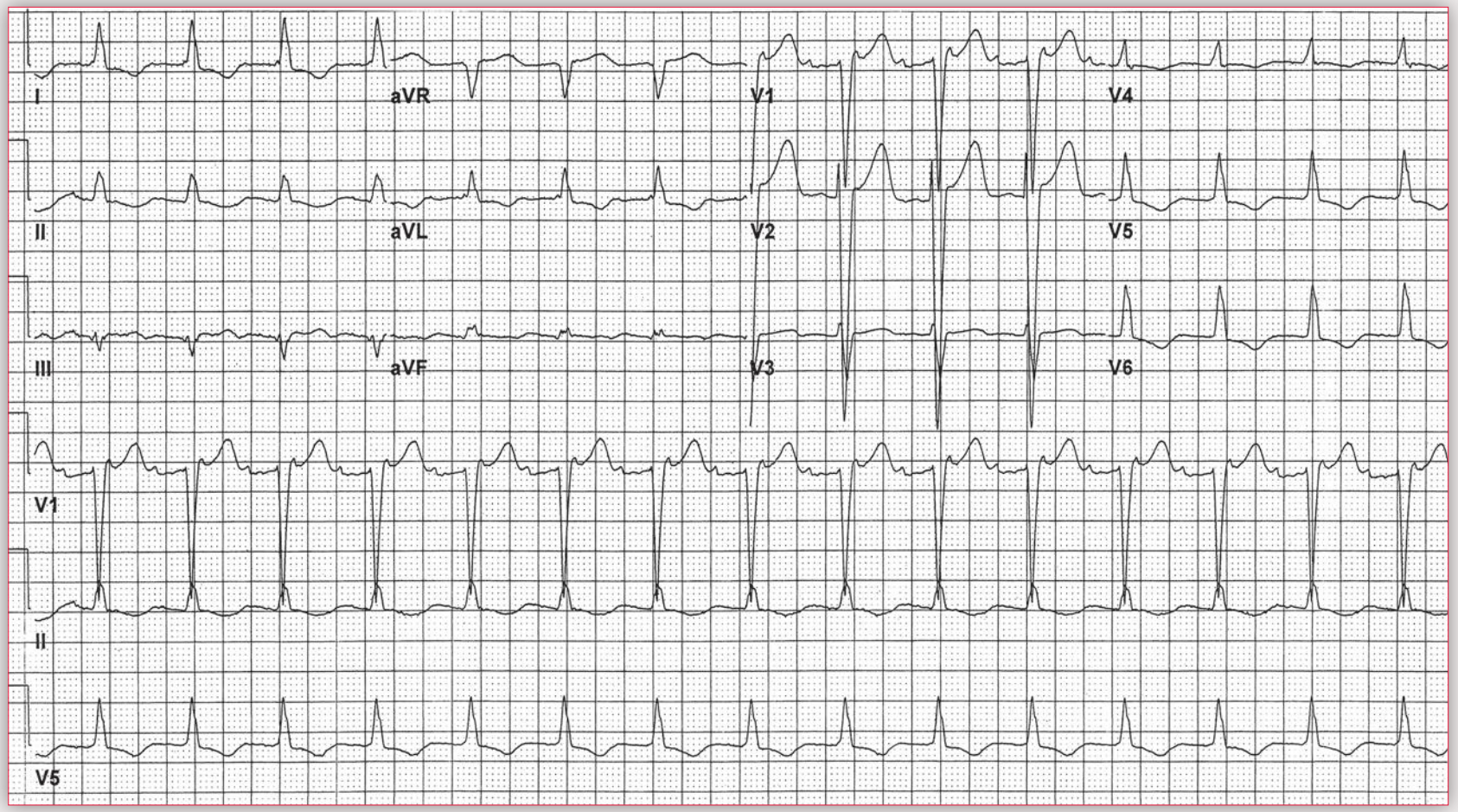
This series of rhythm strips show narrow QRS complexes (0.08 sec) at a regular rate of 76 bpm. The QRS complexes are preceded by a P wave (+) with a stable PR interval (0.20 sec). The QT/QTc interval is normal (360/405 msec). Noted are episodes of a nonsustained wide QRS complex tachycardia (↔) with variability of QRS morphology and axis. This is polymorphic ventricular tachycardia.

In the presence of a normal QT interval, polymorphic ventricular tachycardia is most often a manifestation of ischemia. The ischemia may be the result of epicardial coronary artery stenosis or subendocardial ischemia as may occur with left ventricular (or possibly right ventricular) hypertrophy. Other causes for myocardial ischemia can also be associated with polymorphic ventricular tachycardia. These include coronary artery anomalies, vasospasm, fibrosis, embolism, and dissection (primary or secondary to an aortic dissection). There are other less common causes for myocardial ischemia, such as a left atrial myxoma or a massive pulmonary embolism. Noncardiac causes include anemia and hypoxemia (of any cause). A rare cause of polymorphic ventricular tachycardia with a normal QT interval is a congenital condition known as catecholaminergic polymorphic ventricular tachycardia. This is a condition due to an abnormality of a ryanodine or calsequestrin gene. ■

Practice Case 76

An 88-year-old man on digoxin presents to his cardiologist for routine follow-up. He is complaining of occasional palpitations and shortness of breath, and an ECG is obtained

ECG 76A



Practice Case 76

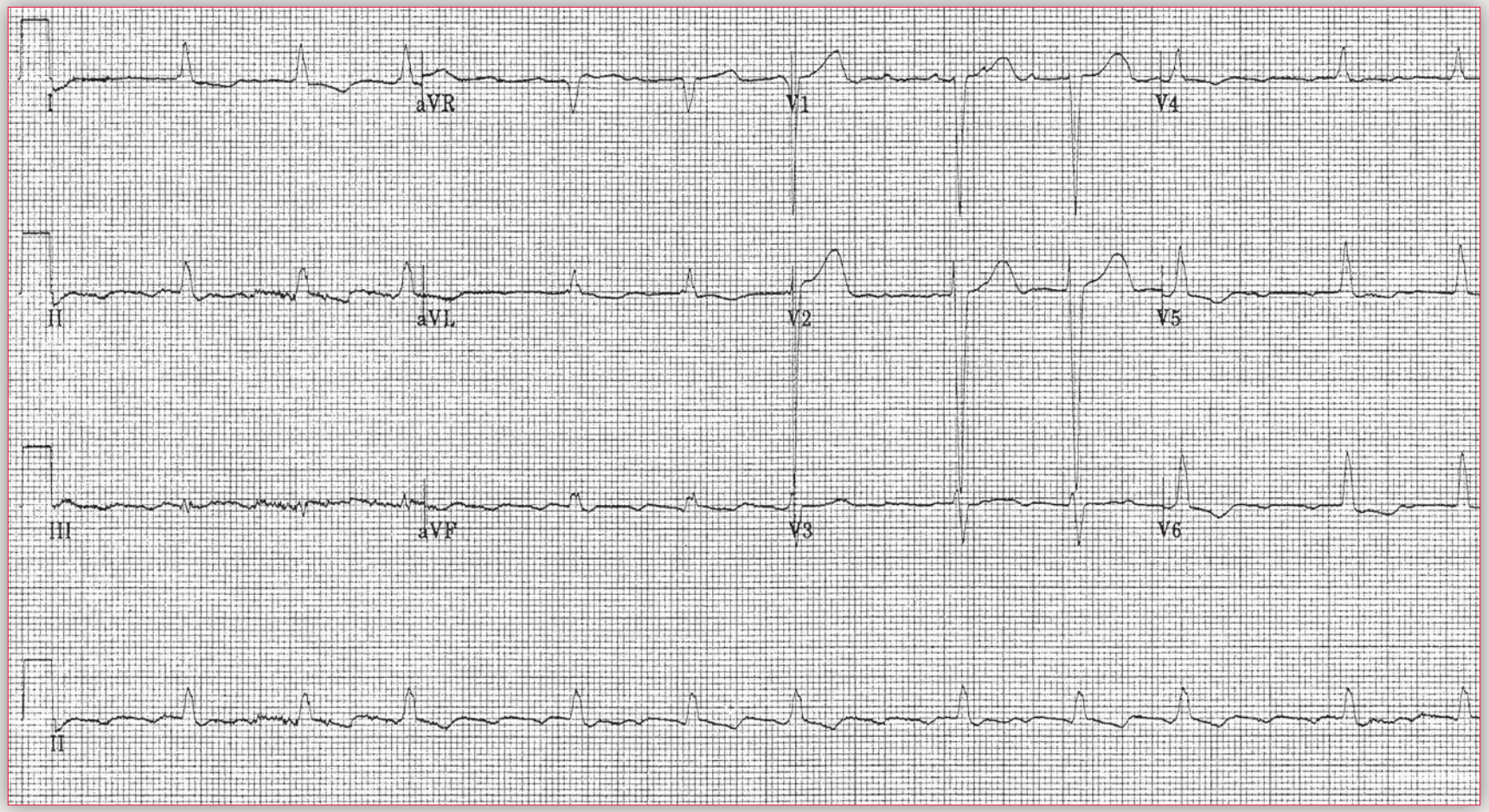
(ECG 76A). The patient is sent to the emergency department for further evaluation, where an intravenous β-blocker is administered. Ten minutes later another ECG is obtained (ECG 76B).

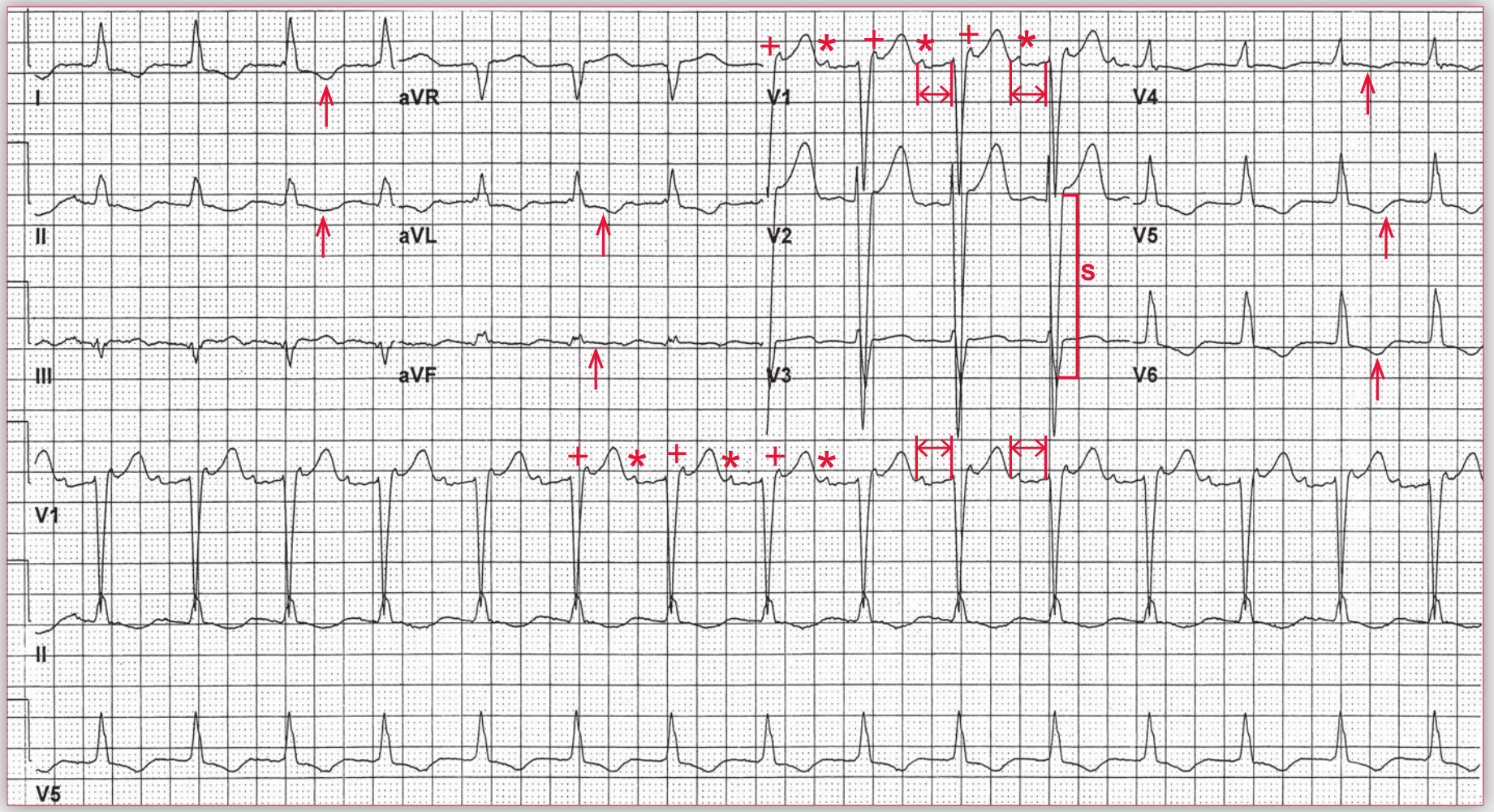
What is the arrhythmia?

What is a possible etiology?

What test should his cardiologist order next?

ECG 76B





ECG 76A Analysis: Atrial tachycardia with 2:1 block, intraventricular conduction delay, left ventricular hypertrophy (LVH)

In **ECG 76A** the rhythm is regular at a rate of 90 bpm. Evidence of atrial activity can be seen, primarily in lead V1 (*). The atrial rate is regular and the PR interval (↔) is constant (0.28 sec). Although this appears to be a sinus rhythm with a first-degree AV block, there is an abnormality noted at the end of the QRS complex in lead V1 (+), which looks like an R′ waveform but has a morphology similar to the obvious P wave. In addition, the interval between the P waves and this waveform at the end of the QRS complex is regular, at a rate of 180 bpm. Although atrial activity is not seen in the other leads, the most likely diagnosis is atrial tachycardia with 2:1 AV block. The QRS complex duration is prolonged (0.12 sec) without any specific pattern; hence this is an intraventricular conduction delay.

The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/440 and 340/415 msec when the prolonged QRS complex duration is considered). The amplitude of the QRS complex is increased, particularly the S wave in lead V2 (30 mm [ ]), which satisfies one of the criteria for left ventricular hypertrophy (LVH, *ie*, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). There are ST-T wave changes (↑) in leads I, aVL, aVF, and V3-V6, possibly associated with LVH.

Atrial tachycardia is not a common arrhythmia and may be associated with digoxin toxicity, especially when there is AV block (*ie*, atrial tachycardia with block). In this case the atrial tachycardia is the result of triggered activity due to delayed after-depolarizations (low-amplitude oscillations resulting from calcium fluxes occurring after phase 3 and at the very beginning of phase 4 of the action potential) and enhanced outputs from the central sympathetic nervous system, which can augment the delayed after-depolarizations resulting in spontaneous action potentials that can cause atrial tachycardia. Digoxin also causes an increase in peripheral vagal tone that slows and blocks conduction through the AV node. A digoxin level should be obtained and digoxin withheld. However, other causes of atrial tachycardia include cardiomyopathy with heart failure, pulmonary disease, myocardial infarction, alcohol excess, hypokalemia, hypoxia, sympathomimetic agents, and cocaine. In these situations AV block may be the result of underlying AV nodal disease.

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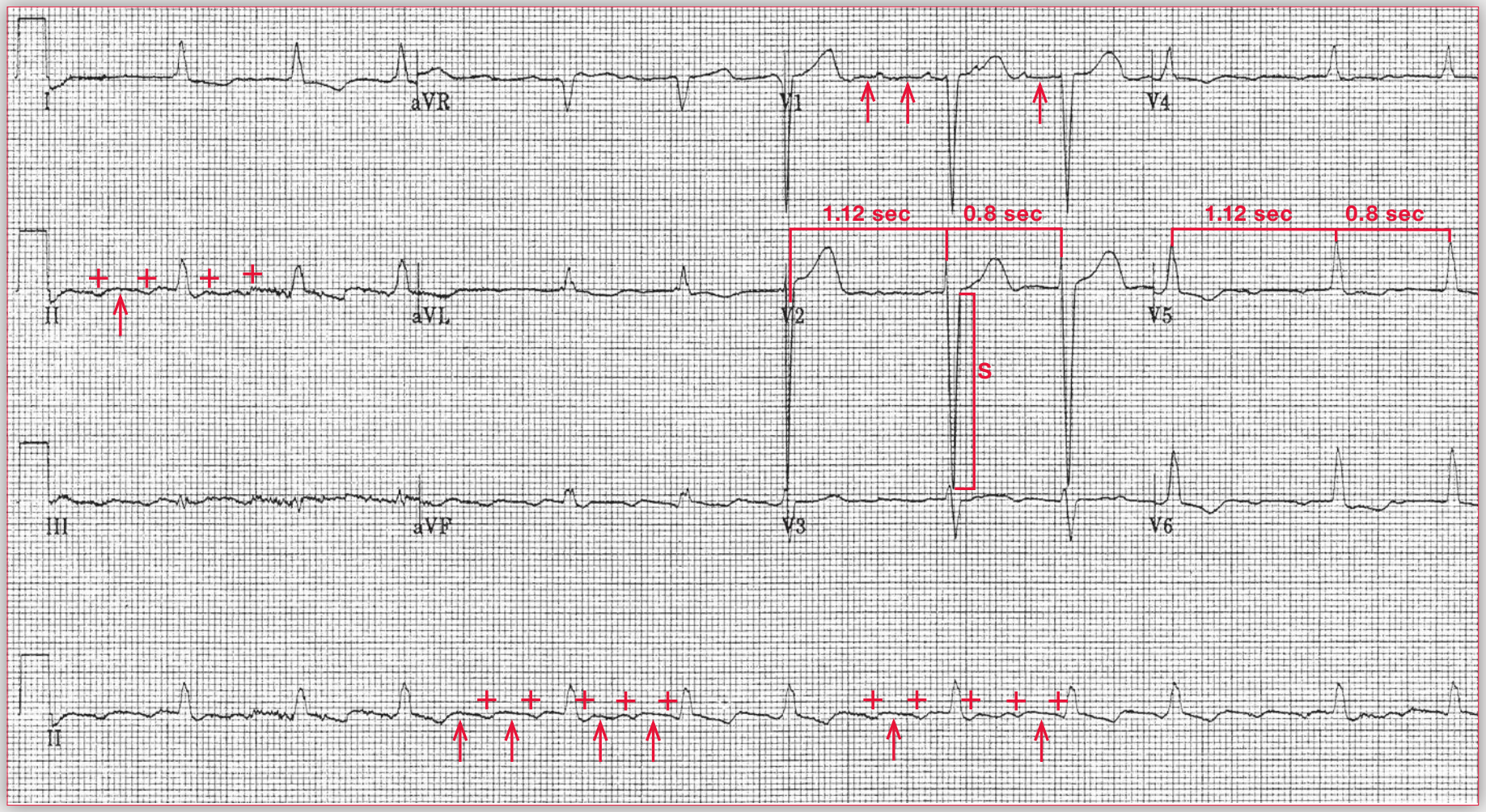


ECG 76B Analysis: Atrial tachycardia with variable block, intraventricular conduction delay, LVH

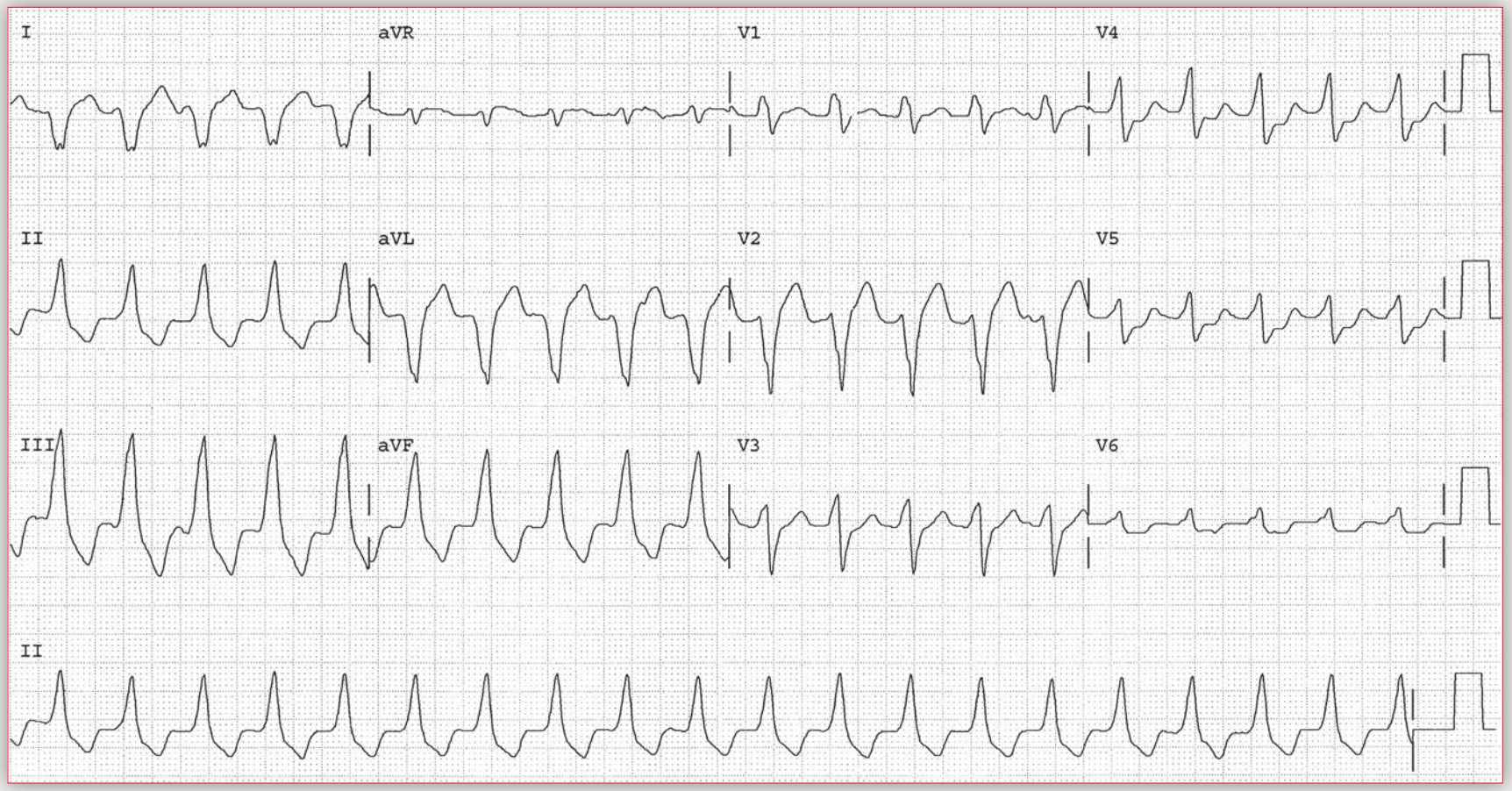
The QRS complex duration, morphology, and axis in **ECG 76B** are the same as in **ECG 76A**. The rhythm is irregular, but there is a pattern seen as all the long RR intervals are the same (1.12 sec) and all the short RR intervals are the same (0.8 sec). Hence the rhythm is regularly irregular. Regular atrial activity (**+**) can be seen at a rate of 180 bpm (identical to the atrial rate seen in **ECG 76A**) and the P waves are negative or inverted in leads II, aVF, and V5-V6. There is an isoelectric baseline (↑) between the P waves, and hence this is atrial tachycardia.

The irregularity of the RR intervals is the result of variable AV conduction (*ie*, 4:1, 3:1, and 2:1). Also noted is variability of the PR intervals as a result of antegrade concealed conduction. When the AV node is stimulated at a rapid rate some impulses get through and some are completely blocked, but others may partially penetrate the AV node. These impulses are not completely conducted through the AV node but are extinguished (concealed) within the AV node, resulting in partial AV nodal depolarization (*ie*, concealed conduction) and prolongation of the refractory period. A subsequent atrial impulse may get through the AV node; however, as there is a prolongation of AV nodal refractoriness, the rate of conduction through the AV node of a subsequent impulse will be slower. ■

Practice Case 77

A 36-year-old man presents with acute onset of palpitations. He has no known history of heart disease. You obtain an ECG (77A) when the patient is symptomatic but still has a normal blood pressure. This ECG is compared with the patient's baseline ECG (77B) obtained during a prior visit.

ECG 77A

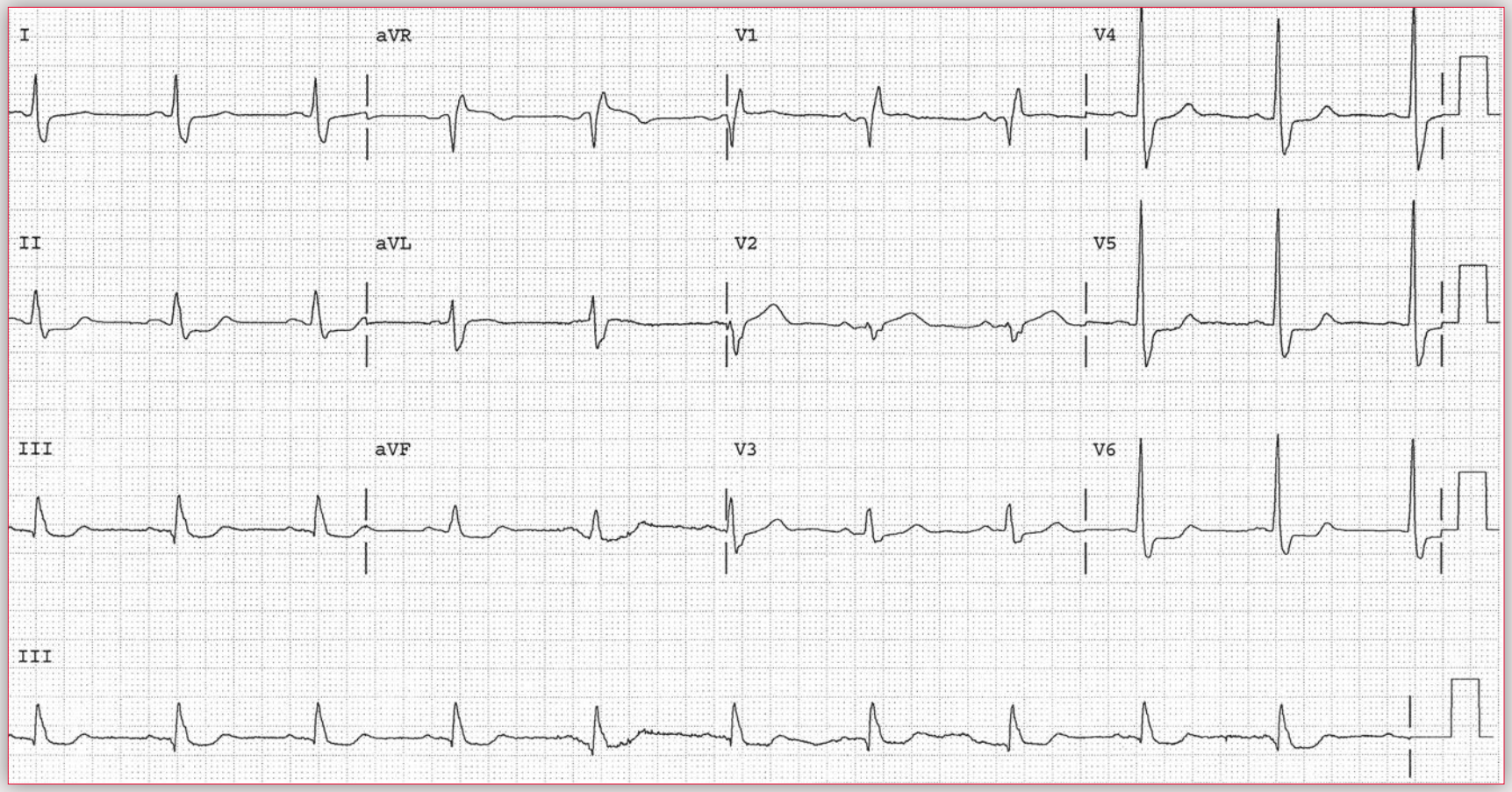


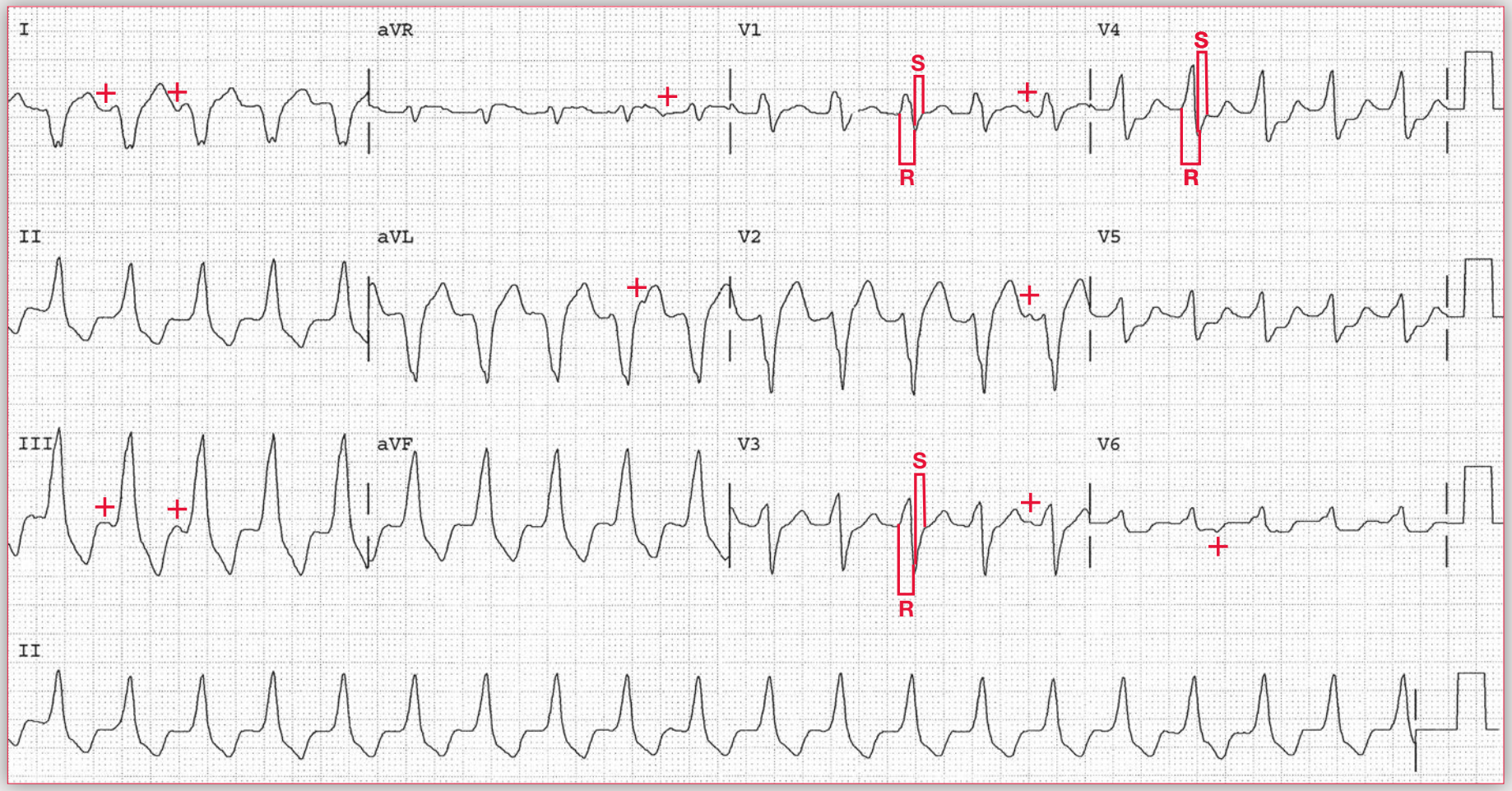
Practice Case 77

What is the origin of the tachyarrhythmia?

A. Sinus node (with aberrant conduction)
B. Ectopic atrial focus (with aberrant conduction)
C. Right ventricle
D. Left ventricle, inferior wall
E. Left ventricle, lateral wall outflow tract

ECG 77B





ECG 77A Analysis: Monomorphic ventricular tachycardia

ECG 77A shows a regular rhythm at a rate of 126 bpm, and the QRS complex duration is increased (0.18 sec). The QRS complex morphology is abnormal (and does not resemble a typical right or left bundle branch block) and the axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are no obvious P waves before or after any of the QRS complexes. However, with careful inspection there are subtle changes in the ST segments (+), which is suggestive of atrial activity or P waves. This is seen particularly in leads I and III just prior to the third QRS complex, just after the fourth QRS complex in lead aVL, and just prior to the fifth QRS complex in leads V1-V3. As P waves are seen before some, but not all, of the QRS complexes, this represents AV dissociation. In association with a wide complex tachycardia with an abnormal QRS complex morphology, AV dissociation is characteristic of ventricular tachycardia. As all the QRS complexes have the same morphology, this is monomorphic ventricular tachycardia. Although the irregularities of the ST segments may not clearly be P waves, such irregularities are seen in ventricular tachycardia and are not seen in supraventricular tachycardia in which there is uniformity of the ST segments as well as QRS complex morphology. This is because activation of the ventricles is via the normal Purkinje system (or an accessory pathway) and the activation sequence (depolarization and repolarization) is the same for each complex.

In addition to AV dissociation, the width of the QRS complex (> 160 msec) is consistent with a diagnosis of ventricular tachycardia. Also noted is the fact that in leads V1, V3, and V4, which have an RS morphology, the R-wave amplitude is greater than the S-wave depth (*ie*, R/S > 1) and the R-wave duration or interval is longer than 100 msec, which means that the initial depolarization of the ventricle is slow and abnormal. This is a feature of a ventricular complex. Wide QRS complexes due to aberration have an S wave that is wider than the R wave is tall (R/S < 1) and an R-wave interval or duration that is less than 100 msec because aberration is generally a delay in the terminal forces of the QRS complex. This is one of the Brugada criteria for distinguishing ventricular tachycardia from supraventricular tachycardia with aberration.

The wide complex tachycardia has a morphology that is suggestive of (but not typical for) right bundle branch block (RBBB) because the monophasic R wave is taller than the S wave is deep in lead V1. The lack of an RSR′ pattern in lead V1 also favors the diagnosis of ventricular tachycardia (instead of supraventricular tachycardia with RBBB).

The diagnosis of ventricular tachycardia excludes answers A and B as the origin of the tachyarrhythmia. Whenever ventricular tachycardia is diagnosed, one can then use the 12-lead ECG to determine a more precise location within the ventricles as the origin (*eg*, to identify the location of myocardial scar or ischemia). An RBBB-like ventricular tachycardia (R wave taller than S-wave depth in lead V1) typically originates from the left ventricle; a left bundle branch block–like ventricular tachycardia generally originates from the right ventricle. The QRS complex leads that are most negative in deflection represent the source of the ventricular tachycardia (*ie*, the depolarization vector

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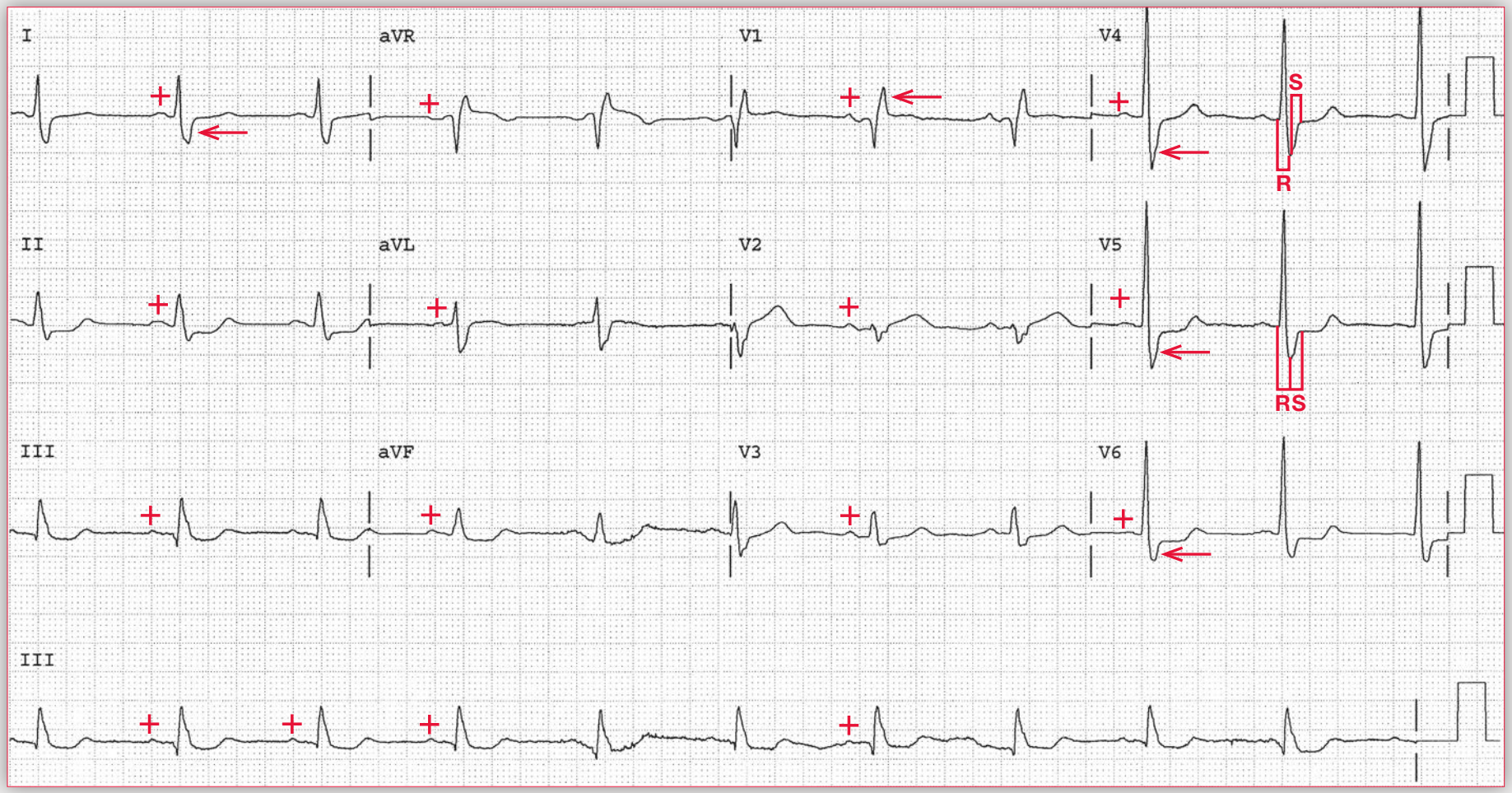


ECG 77B Analysis: Normal sinus rhythm, right bundle branch block

points away from the origin). In ECG **77A**, the ventricular tachycardia has an RBBB-like morphology (R wave taller than S-wave depth in lead V1) and thus the ventricular tachycardia comes from the left ventricle. Leads I and aVL are negative, implicating the left ventricular lateral wall as the origin. Because the QRS complexes in leads II, III, and aVF are positive (inferiorly directed), the depolarization vector starts at the outflow tract or base of the heart and moves toward the inferior wall. Hence, this is lateral left ventricular outflow tract (LVOT) tachycardia (answer E). LVOT tachycardias are a form of a repetitive monomorphic ventricular tachycardia and are similar to right ventricular outflow tract tachycardias. LVOT tachycardias usually occur in individuals with a structurally normal heart, tend to be well tolerated hemodynamically, are not associated with sudden cardiac arrest, and are often responsive to verapamil or β-blocker therapy.

ECG 77B shows a regular rhythm at a rate of 62 bpm. There is a P wave (**+**) before each QRS complex with a stable PR interval (0.18 sec). The P wave is upright in leads I, II, aVF, and V4-V6; hence this is a sinus rhythm. The QRS complex duration is increased (0.14 sec) and the morphology is typical of an RBBB; that is, there is a broad S wave in leads I and V5-V6 (←) and a broad terminal R wave in lead V1 (→) (a qR morphology). Note that the widened QRS complex is the result of terminal delay; that is, it is the terminal portion of the QRS complex that accounts for the widening (*ie*, in leads with an RS complex, the S wave is wider than the R wave, or R/S < 1, and the R-wave duration is < 100 msec). Comparison with the QRS complex in **ECG 77A** clearly demonstrates major differences in morphology. The QT/QTc intervals are prolonged (480/490 msec) but normal when corrected for the prolonged QRS complex duration (440/450 msec). ■

Notes

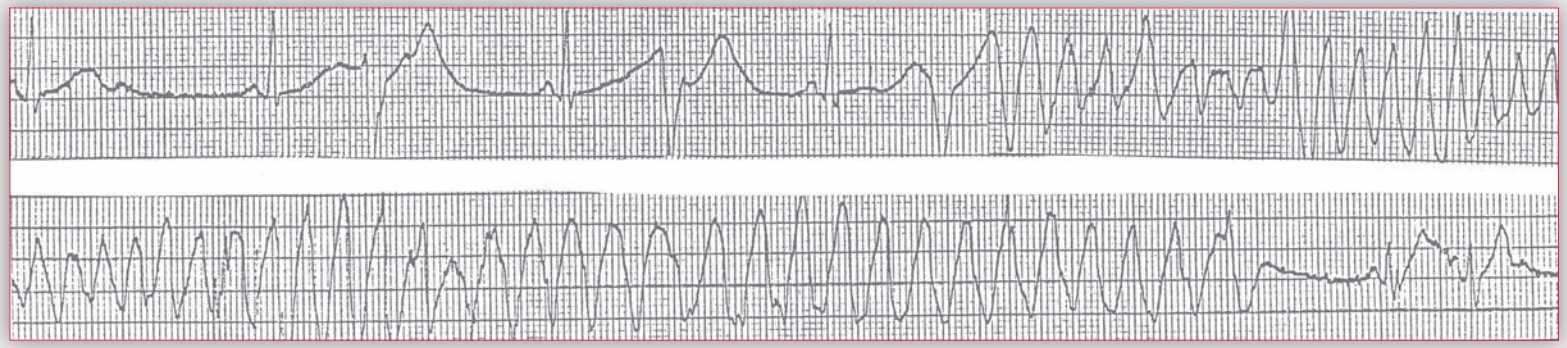
Practice Case 78

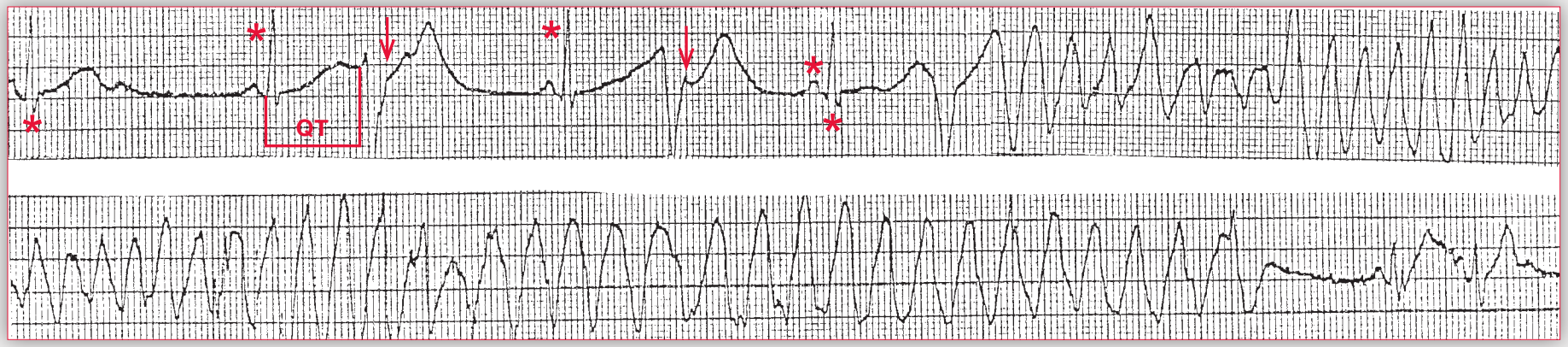
A 62-year-old woman with a history of hypertension for which she takes atenolol presents with 4 days of diarrhea, nausea, and vomiting. Her primary care physician started her on levofloxacin 2 days ago for the diarrhea, but stool cultures have thus far been negative. The patient reports extremely poor oral intake due to the nausea, but she has managed to take her atenolol every day. While in the emergency department, she is placed on telemetry and receives ondansetron for her nausea. Initial laboratory analysis reveals a serum potassium level of 2.2 mmol/L (normal range, 3.5–5.0 mmol/L) and pre-renal azotemia with creatinine elevated to 1.9 mg/dL from her baseline of 0.7 mg/dL. She suffers acutely from a brief syncopal episode (loss of consciousness for 10 to 15 seconds) and has the following associated rhythm strip.

What is the mechanism of this arrhythmia?

What are the clinical factors contributing to this arrhythmia?

How would you manage this patient?





ECG 78 Analysis: Nonsustained polymorphic ventricular tachycardia with QT prolongation (torsade de pointes), sinus bradycardia, premature ventricular complexes, long QT interval

This is a continuous lead II tracing from a telemetry monitor. The first, second, fourth, and sixth QRS complexes (*) have a normal duration (0.08 sec), and they are preceded by a P wave (+) with a stable PR interval (0.16 sec). The third and fifth QRS complexes (↓) are early and wide without a preceding P wave; these are premature ventricular complexes (PVCs). After the sixth QRS complex, there is a wide QRS complex tachycardia and the QRS complexes have variable morphologies and changing axis. This is polymorphic ventricular tachycardia. Although the PVCs interrupt the QT interval, it can be seen that the QT interval is at least 640 to 680 msec and is therefore markedly prolonged. Although the T wave is interrupted, the QT interval can be estimated by continuing the slope of the T wave until it crosses the baseline. When the baseline QT interval of a sinus complex is prolonged, the polymorphic ventricular tachycardia is termed torsade de pointes (twisting of points).

The mechanism of torsade is often referred to as "R on T," which signifies a PVC ("R") coming in shortly after the apex of the T wave, which represents the vulnerable period. This is at the very end of ventricular repolarization. The likelihood of "R on T" increases as the QT interval prolongs. One of the leading pathophysiologic hypotheses is that there are derangements in cardiac ion flow during phases 2 and 3 of the action potential, primarily a block of potassium ion efflux, leading to prolongation in the membrane-refractory period and an increase in the action potential duration. During the prolonged action potential there is a potential for the development of early after-depolarizations, a form of triggered activity. Early after-depolarizations may be the result of calcium fluxes, which are enhanced during the prolongation of phase 2 of the action potential. The development of acquired QT prolongation is related to a long list of medications and may be potentiated by bradycardia, hypokalemia, or hypomagnesemia. The occurrence of torsade is related to the repeated triggered activity that develops as a result of the prolongation of phase 2 of the action potential, allowing for the development of early after-depolarizations.

This patient has a clinical history suggestive of acquired QT prolongation. She has received two medications that prolong the QT interval, a quinolone and ondansetron. Additionally, due to her gastrointestinal illness, vomiting, and poor oral intake, she is hypokalemic. Such electrolyte disturbances can cause QT prolongation as well as induce ventricular ectopy. In addition to these clinical factors, bradycardia is known to be associated with an increased risk for drug-induced torsade because the QT interval (membrane refractory period) prolongs further with bradycardia and shortens with tachycardia. In addition, drug-induced torsade may be "pause dependent"; that is, it may occur following a long RR interval (with resultant further prolongation of the ventricular refractory period) as happens with PVCs, which are often followed by a compensatory pause. The patient has underlying sinus bradycardia, likely from elevated levels of atenolol, which is renally cleared, as well as a PVC with an associated pause.

continues

The management of torsade de pointes begins with an evaluation of the patient's vital signs and level of consciousness with focus on maintaining airway, breathing, and circulation. If torsade is sustained and the patient is unstable, immediate defibrillation is warranted. Medical treatment to suppress the occurrence of torsade includes the immediate administration of intravenous magnesium, which stabilizes the myocardium and may work by interfering with the influx of calcium ions during phase 2 of the action potential, thereby reducing early afterdepolarizations. Additional therapy to prevent recurrent episodes of torsade focuses on normalization of the QT interval and prevention of bradycardia. This includes:

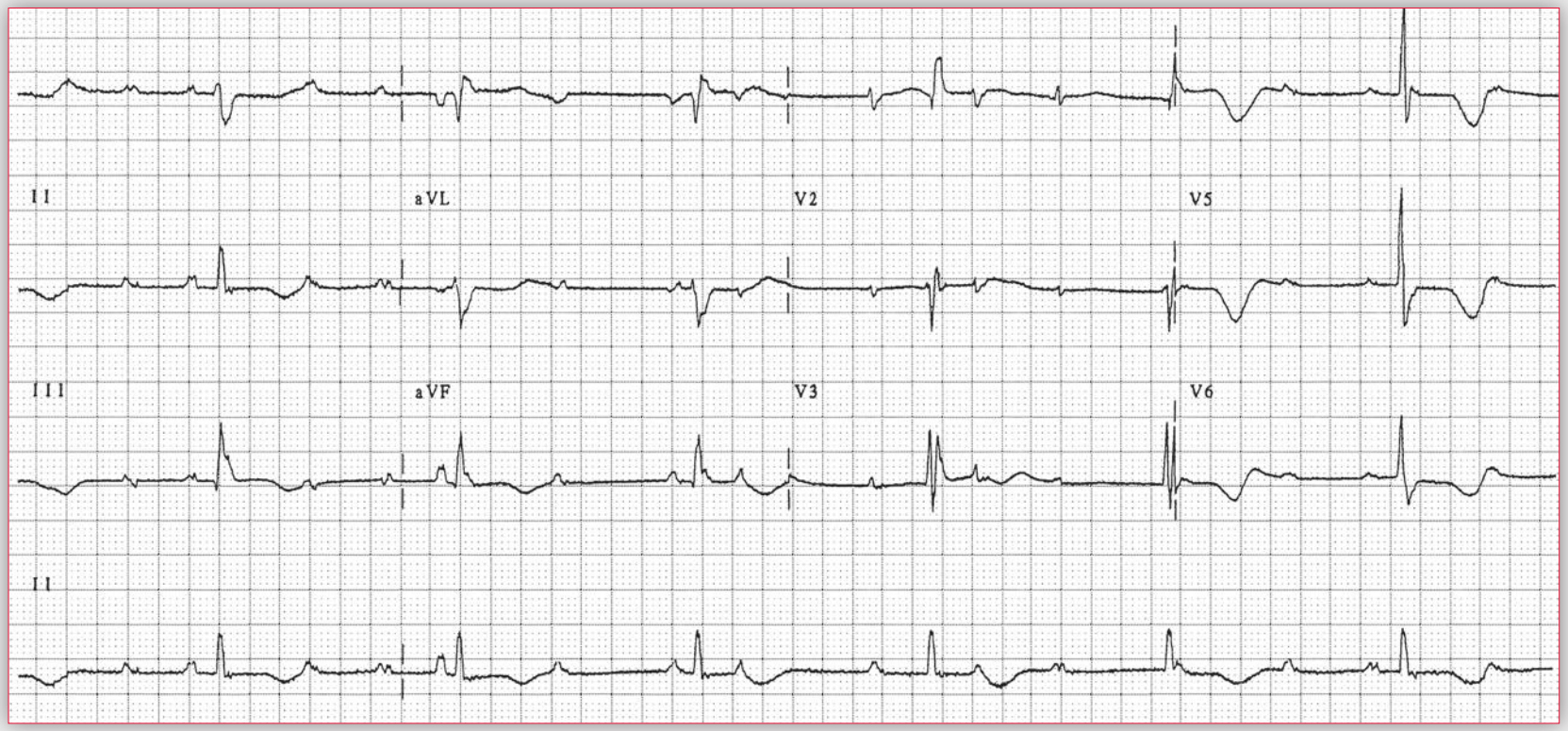
- Correcting any electrolyte abnormalities
- Avoiding all QT prolonging agents
- Administering intravenous lidocaine (class Ib antiarrhythmic)
- Withholding any nodal blocking agents, especially β-blockers, to avoid bradycardia
- Increasing the heart rate, which will shorten the membrane-refractory period and the action potential duration, with either electronic pacing or intravenous isoproterenol (a β-agonist). Pacing at a sufficient heart rate may also suppress premature ventricular complexes, which may be a trigger for torsade. ■

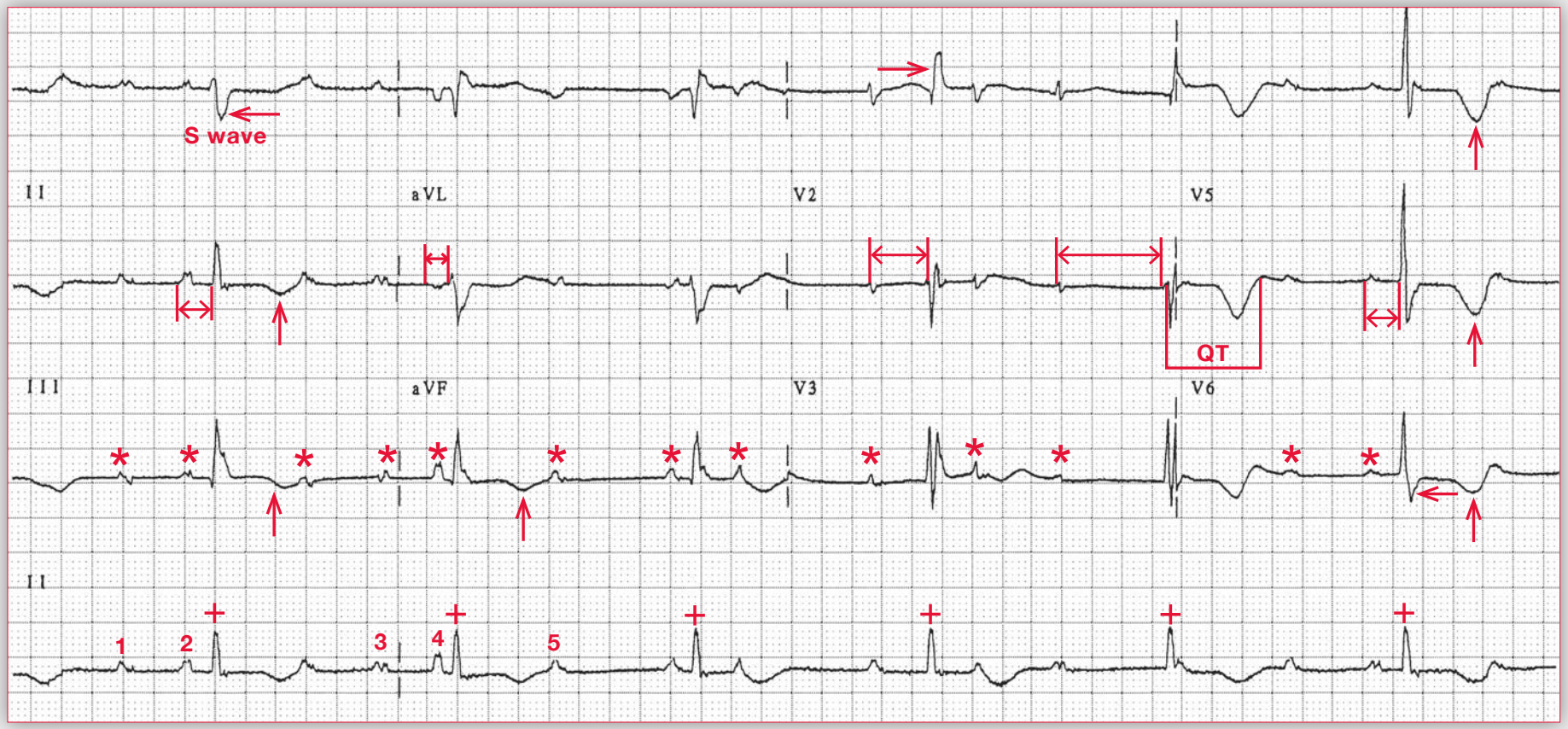
Practice Case 79

A 68-year-old man presents via emergency medical services to the emergency department with a syncopal episode as well as the acute onset of chest pain and shortness of breath. His medical history, obtained from his wife, includes hypertension and paroxysmal atrial fibrillation. Medications include an angiotensin-converting enzyme inhibitor, a β-blocker, and verapamil. He is awake and alert in the emergency department and complains of acute-onset dyspnea and lightheadedness that began just prior to his activating emergency services. He appears to be in moderate distress. You note that his radial pulse is slow and regular. His blood pressure is 85/30 mm Hg by sphygmomanometer. His oxygen saturation is 86% by pulse oximetry on ambient air, and improves with supplemental oxygen via nasal cannula. His physical exam shows jugular venous pressure of 14 cm H_2O with intermittent cannon A waves, clear lung fields with subtle wheezing over the posterior right base, a nondisplaced left ventricular point of maximal impulse, an absence of lifts and murmurs, a normal abdomen, and palpable extremity pulses. Asymmetric left lower extremity edema is noted. The emergency department nurse has obtained an ECG and presents it to you. A baseline ECG is not available.

What is the diagnosis?

What is the immediate management?





ECG 79 Analysis: Wandering atrial pacemaker, complete heart block with junctional escape rhythm, right bundle branch block, right axis due to left posterior fascicular block

The QRS complexes are regular (+), at a rate of 40 bpm. However, there is evidence of atrial activity (*) at an average rate of 90 bpm; the atrial activity is irregularly irregular. There are at least three P-wave morphologies with no one P-wave morphology being dominant. Hence the underlying atrial rhythm is a wandering atrial pacemaker or multifocal atrial rhythm. However, there is a regular ventricular rate of 40 bpm. Wandering atrial pacemaker is associated with a rhythm that is irregularly irregular, as ventricular activation results from the irregularly irregular atrial activity. In addition, noted is the fact that there is no relationship between the P waves and the QRS complexes; that is, there is a variable PR interval (↔). Along with the regularity of the QRS complexes, this means that there is AV dissociation. As the atrial rate is faster than the ventricular rate, this is complete heart block. The QRS complexes are wide (0.16 sec), and they have a right bundle branch block (RBBB) morphology (qR morphology in lead V1 [→] and broad S wave in leads I and V6 [←]) as well as a right axis, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF); this is consistent with a left posterior fascicular block as there are no other etiologies for the right axis suggested by the ECG. It should be noted that the QRS complex in lead I is still negative even when the S wave (reflecting the RBBB) is ignored. Hence this is a wandering atrial pacemaker with complete heart block and an escape junctional rhythm. There are also nonspecific T-wave abnormalities (↑). The QT/QTc intervals are slightly prolonged (600/490 msec). However, when considering the widened QRS complex, the QT/QTc intervals are normal (540/440 msec).

This patient's presentation is very suspicious for acute pulmonary embolus. The history of acute dyspnea, lightheadedness attributable to hypotension, focal pulmonary wheezes, and asymmetric lower extremity edema is consistent with this diagnosis. His ECG shows a wandering atrial pacemaker (or multifocal atrial rhythm), probably the result of acute right atrial stretch caused by the abrupt rise in right atrial pressure seen with pulmonary embolism. Similarly, stretch

continues

of the right ventricle, when extreme, may result in stretch injury of the right bundle branch, whose proximal fibers run in the interventricular septum. This manifests as an acute RBBB on the surface ECG. It is uncertain whether the RBBB is chronic or related to possible acute right ventricular stretch. A left posterior fascicular (hemi) block is an uncommon conduction abnormality as the left posterior fascicle (a branch of the left bundle) is a fanlike structure that covers a large portion of the inferior aspect of the left ventricle. It may be seen in cardiomyopathies that affect this region of the left ventricle, especially associated with a prior myocardial infarction. Criteria for a left posterior fascicular block are met when a right axis is seen with a normal QRS width (unless accompanied by an RBBB). Other causes of right axis, such as right ventricular hypertrophy from any cause, lateral wall myocardial infarction, right–left arm lead switch, Wolff-Parkinson-White pattern, and dextrocardia, should be excluded before left posterior fascicular block is diagnosed; that is, left posterior fascicular block is a diagnosis of exclusion. An acute right axis may occur as a result of a massive pulmonary embolism if there is significant elevation in pulmonary artery and right ventricular pressures. This may be suggested by the presenting symptom of syncope as well as the presence of an RBBB, which also indicates an elevation in right ventricular pressure. However, it is possible that the RBBB and left posterior fascicular block predate this presentation. This can

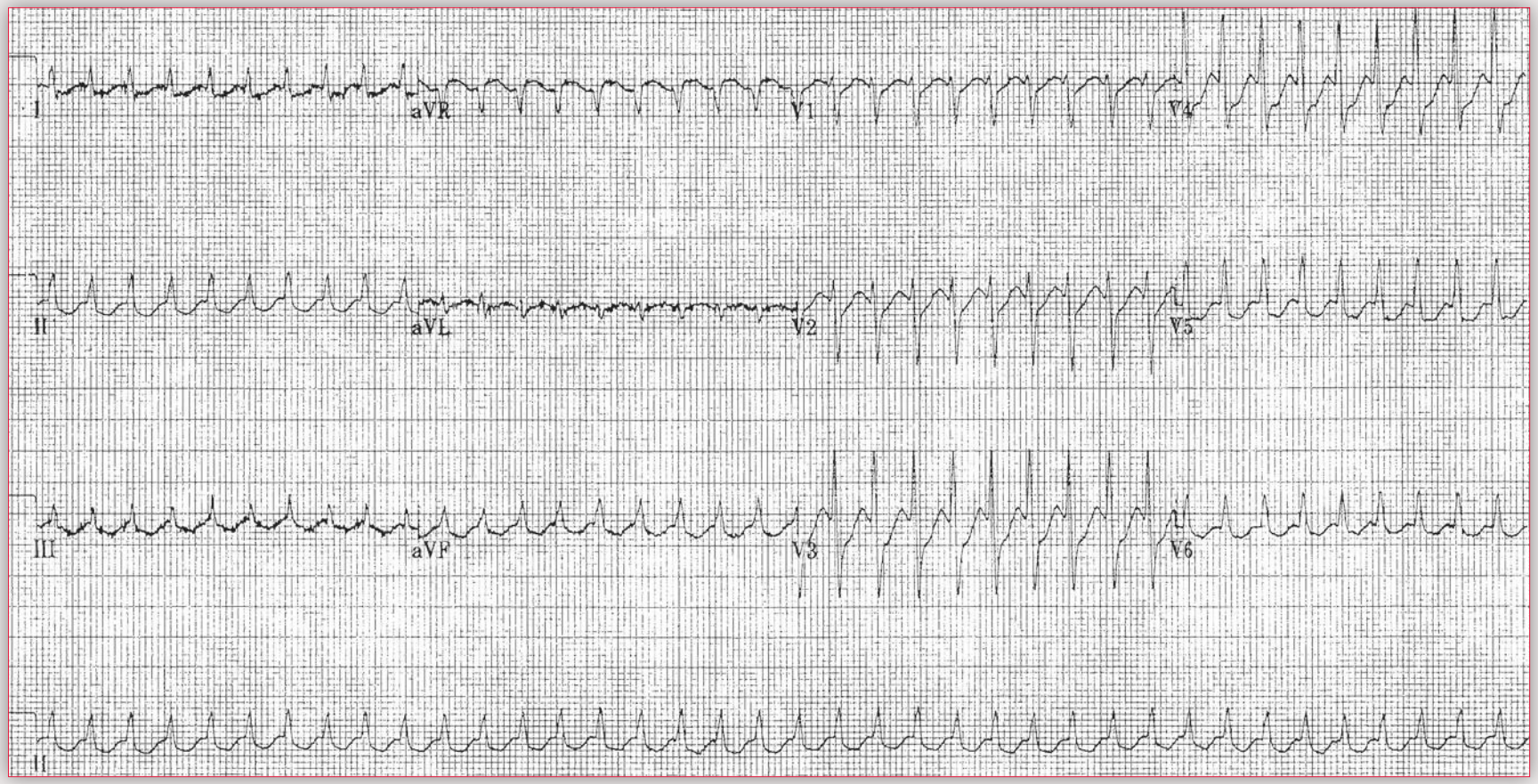
only be established by comparison with a previous ECG. Although there appears to be QT/QTc prolongation, it is important to remember that the QT interval also includes the QRS complex. If there is an increase in the QRS complex duration, as with a bundle branch block, this must be accounted for before considering the QT/QTc intervals. In general, the QRS complex width used for QT/QTc measurement is 80 msec. When the QRS complex is widened, the number of milliseconds greater than 80 is subtracted from the QT interval measurement.

Initial efforts should be made to stabilize the patient with intravenous fluids and confirm the suspected diagnosis (with computed tomography pulmonary angiography). Given his hemodynamic instability, initiation of thrombolytic therapy should be strongly considered if the diagnosis of pulmonary embolism is confirmed. Troponin and brain natriuretic peptide measurements may aid in prognostication. His conduction abnormality may resolve with discontinuation of the β-blocker and verapamil. It may also improve with direct thrombolytic therapy. If the complete heart block and slow ventricular rate persist and there are continued symptoms associated with the bradycardia, placement of a temporary pacemaker wire may be considered. However, use of a pacemaker in the context of acute pulmonary embolism carries a higher procedural risk. ■

Practice Case 80

A 48-year-old man with a history of both atrial fibrillation and atrial flutter is started on flecainide for rhythm control. He is successfully electrically converted to a normal sinus rhythm after initiation of flecainide therapy and does well for several weeks. He then develops

ECG 80A



Practice Case 80

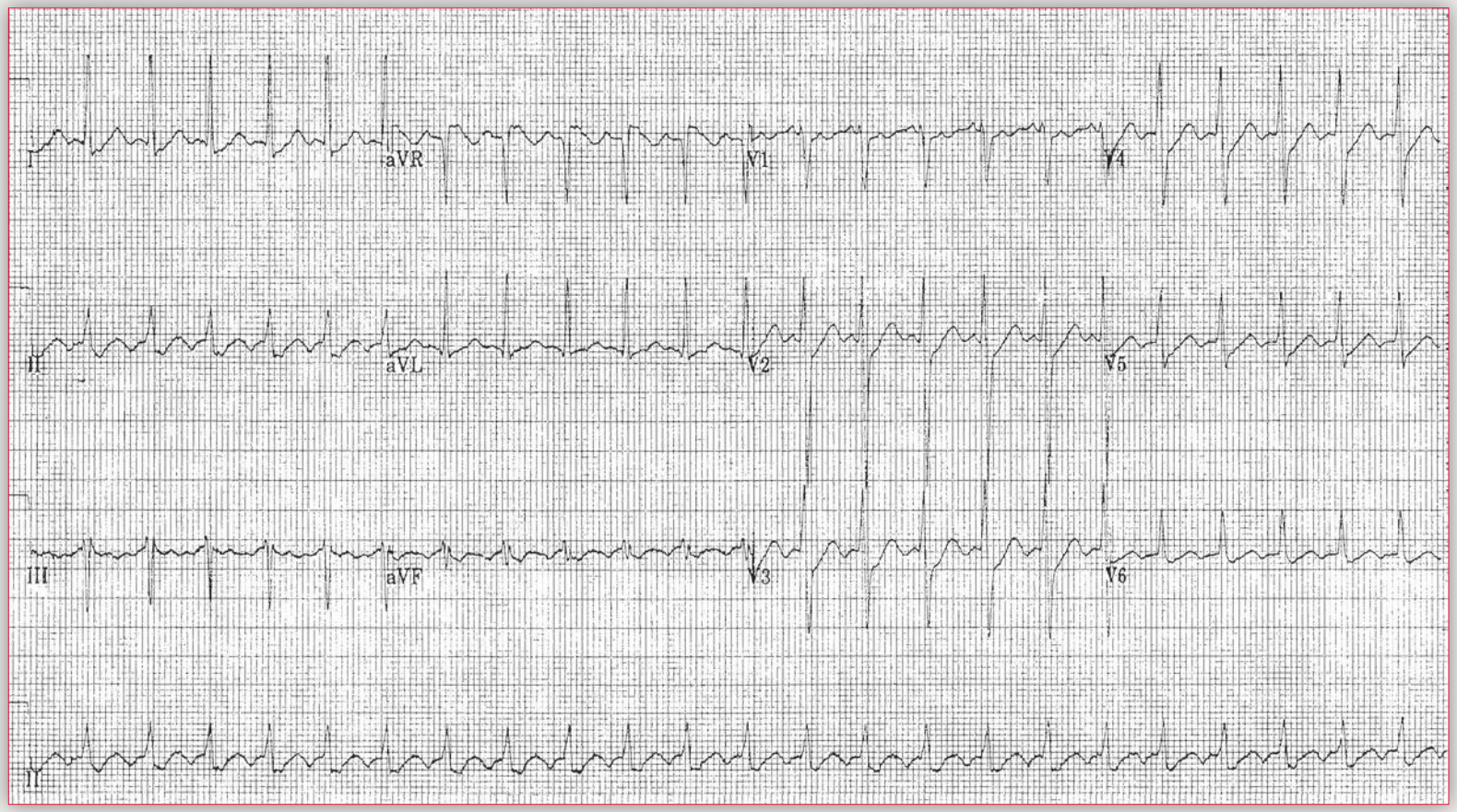
sudden-onset palpitations and pre-syncope. He is brought by emergency medical services to the emergency department, where he is found to be markedly tachycardic. An ECG is obtained (ECG 80A). After therapy is provided to slow his heart rate, a second ECG (80B) is obtained.

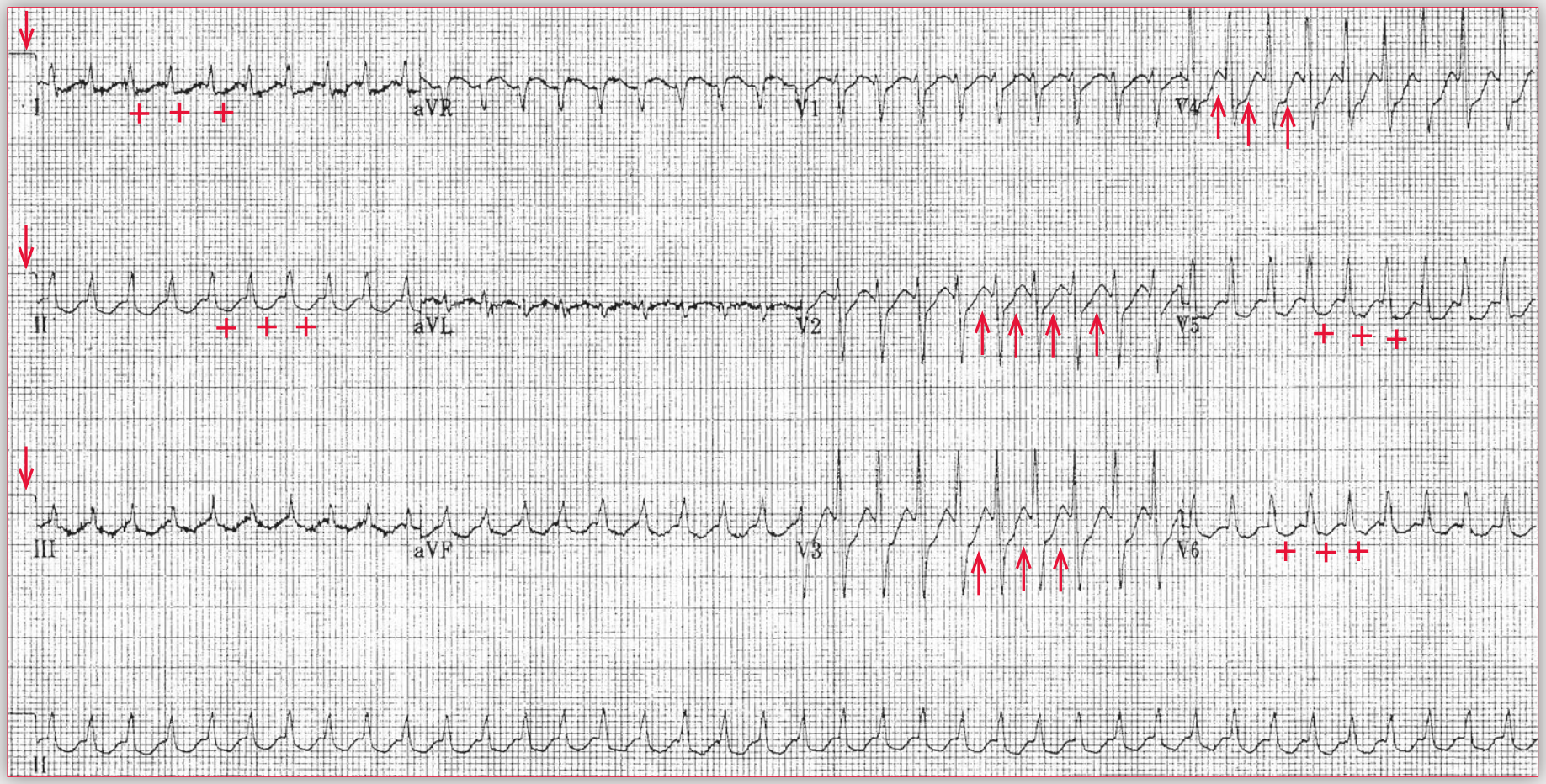
What do the ECGs show?

What accounts for this rapid arrhythmia?

Could it have been prevented?

ECG 80B





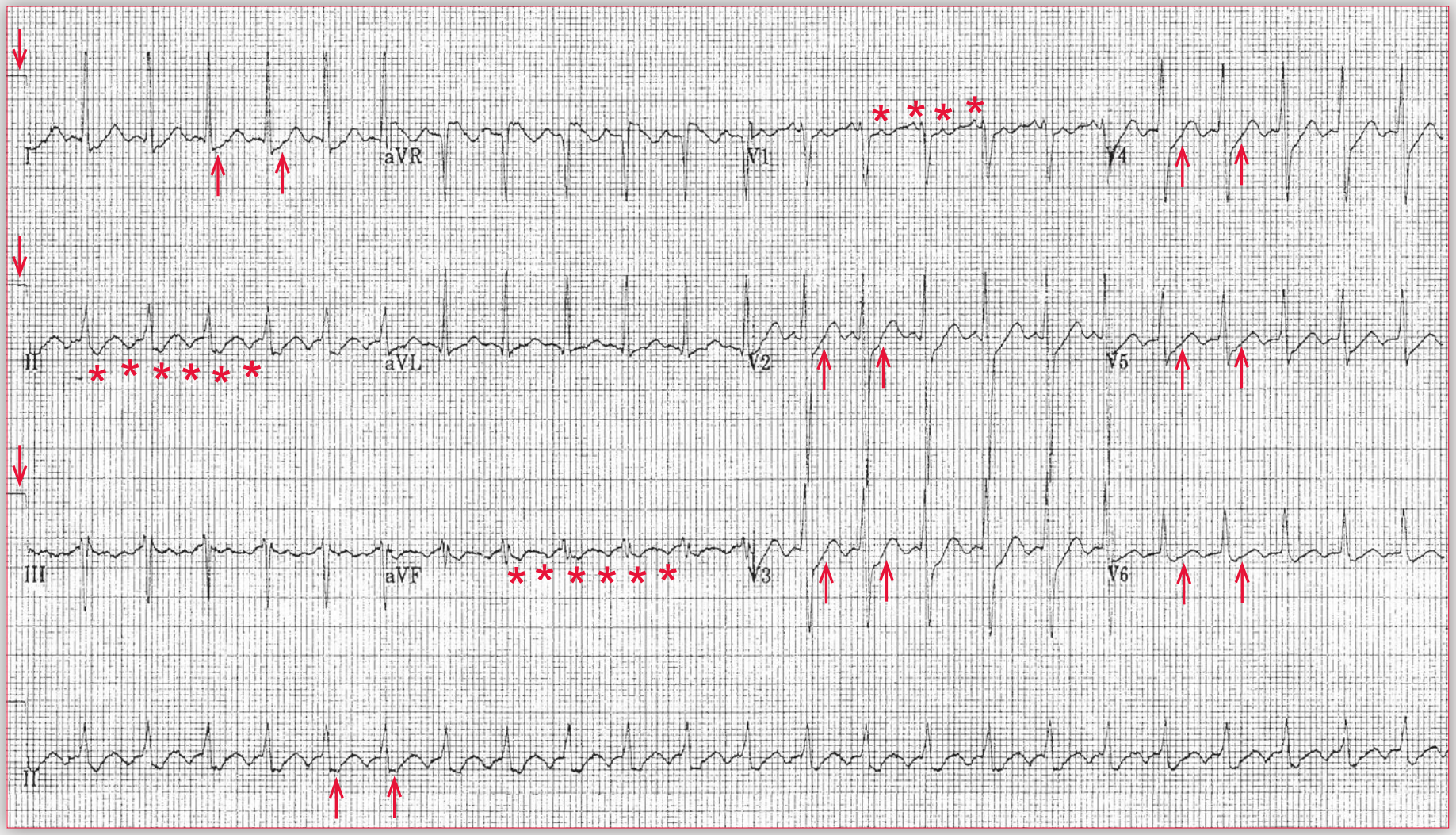
ECG 80A Analysis: 1:1 atrial flutter, low voltage due to recording at half-standard

ECG 80A shows a regular rhythm at a rate of 260 bpm. The QRS complex is of normal duration (0.08 sec); has a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF); and has a normal morphology. The QT/QTc intervals are normal (160/330 msec). This is supraventricular tachyarrhythmia. The only regular supraventricular tachyarrhythmia that presents at an atrial rate of 260 bpm or higher is atrial flutter. When the ventricular rate is also 260 bpm or higher, there is 1:1 AV conduction. Close inspection reveals a suggestion of notching between each RR interval (+) that is likely atrial activity. The voltage is low, particularly in the limb leads (R wave < 5 mm in each limb lead and R wave < 10 mm in each precordial lead). However, it should be noted that the ECG was recorded at half-standard (*ie*, 1 mV = 5 mm, or five small boxes [↓]). Hence the actual amplitude of the waveforms is twice what is measured.

Although the flecainide was successful initially in maintaining normal sinus rhythm, it did not prevent recurrent atrial flutter. Although the initial atrial flutter rate is not known, the rate on this ECG is 260 bpm, which is the lower limit of the rate for atrial flutter. It is possible that the initial flutter rate was faster (*ie*, 300 bpm). Flecainide (and other class 1C and class 1A agents) can slow the flutter rate. If this occurs, there is the potential for 1:1 AV nodal conduction. In addition, with the decrease in the atrial rate there is less concealed conduction; this permits a greater degree of impulse conduction through the AV node. As a result of this effect of class IC (and class 1A) agents, they should always be administered with a β-blocker for producing AV nodal blockade.

Also noted are upsloping ST-segment depressions in leads V1-V4 (↑) and horizontal ST-segment depressions in most of the other leads. Although at this rate the ST-segment depression could represent ischemia, it is also likely that these are actually the flutter waves.

continues



ECG 80B Analysis: Atrial flutter with 2:1 AV conduction

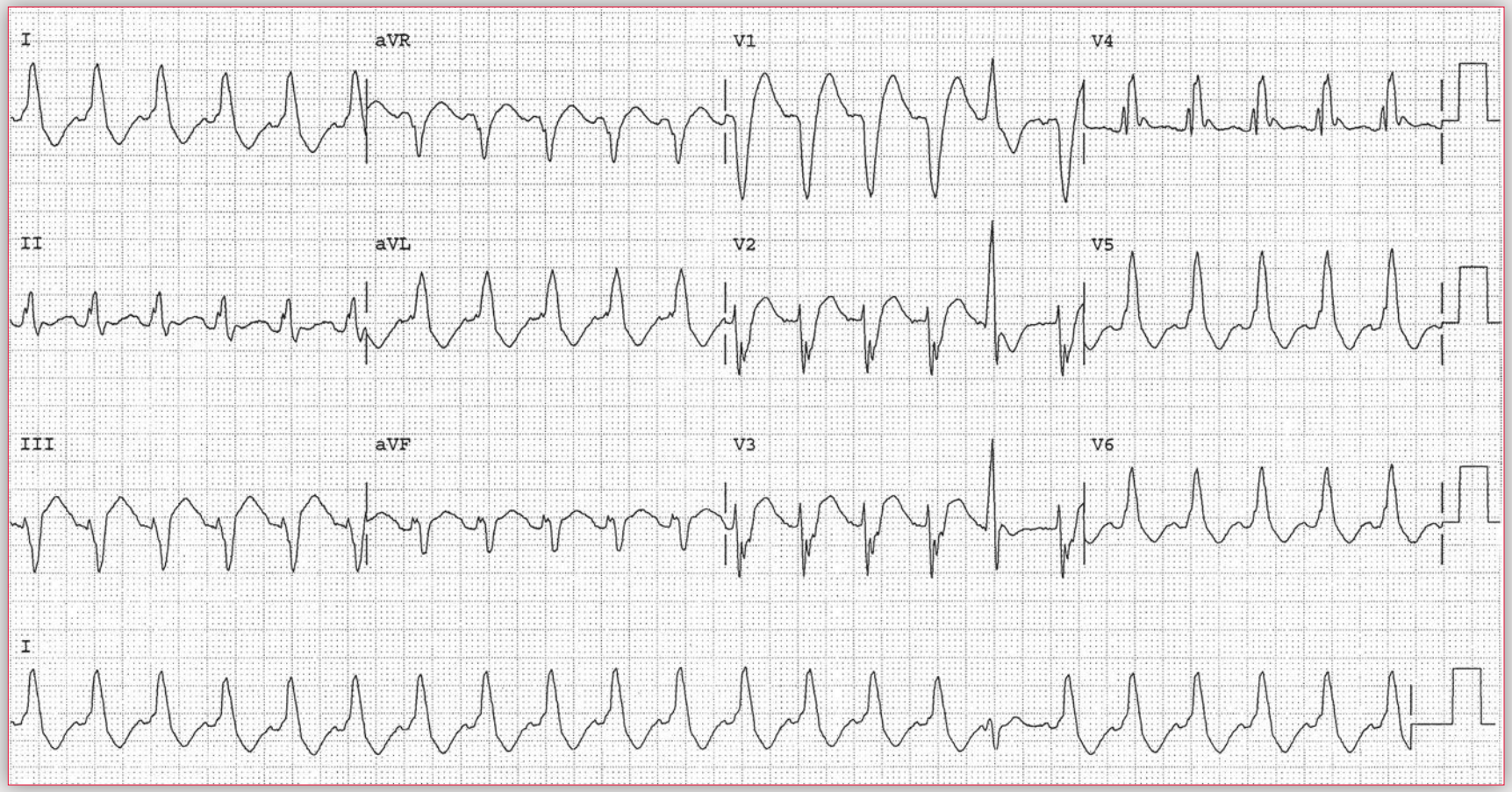
ECG 80B was obtained after the administration of an intravenous β-blocker. The QRS complex duration and morphology are the same as in **ECG 80A**, as are the QT/QTc intervals. The QRS complex amplitude is normal and is higher than in **ECG 80A**. However, it should be noted that this ECG was recorded at normal standard (↓), where 1 mV = 10 mm, accounting for the normal voltage of the QRS complexes. The rhythm is regular at a rate of 130 bpm. Atrial activity (*) with a stable atrial rate of 260 bpm can now be seen, especially in leads II, III, aVF, and V1-V3. Upsloping ST-segment depressions (↑) can be seen in leads I and V2-V6, as well as apparent ST-segment depressions in lead II (↑). However, these changes clearly correlate with the second flutter wave, which is superimposed on the early part of the ST segment. It is common for one of the flutter waves to be superimposed on the initial or terminal portion of the QRS complex and hence resemble a Q wave or an S wave or ST-segment depression. This may make atrial flutter a difficult rhythm to diagnose. ■

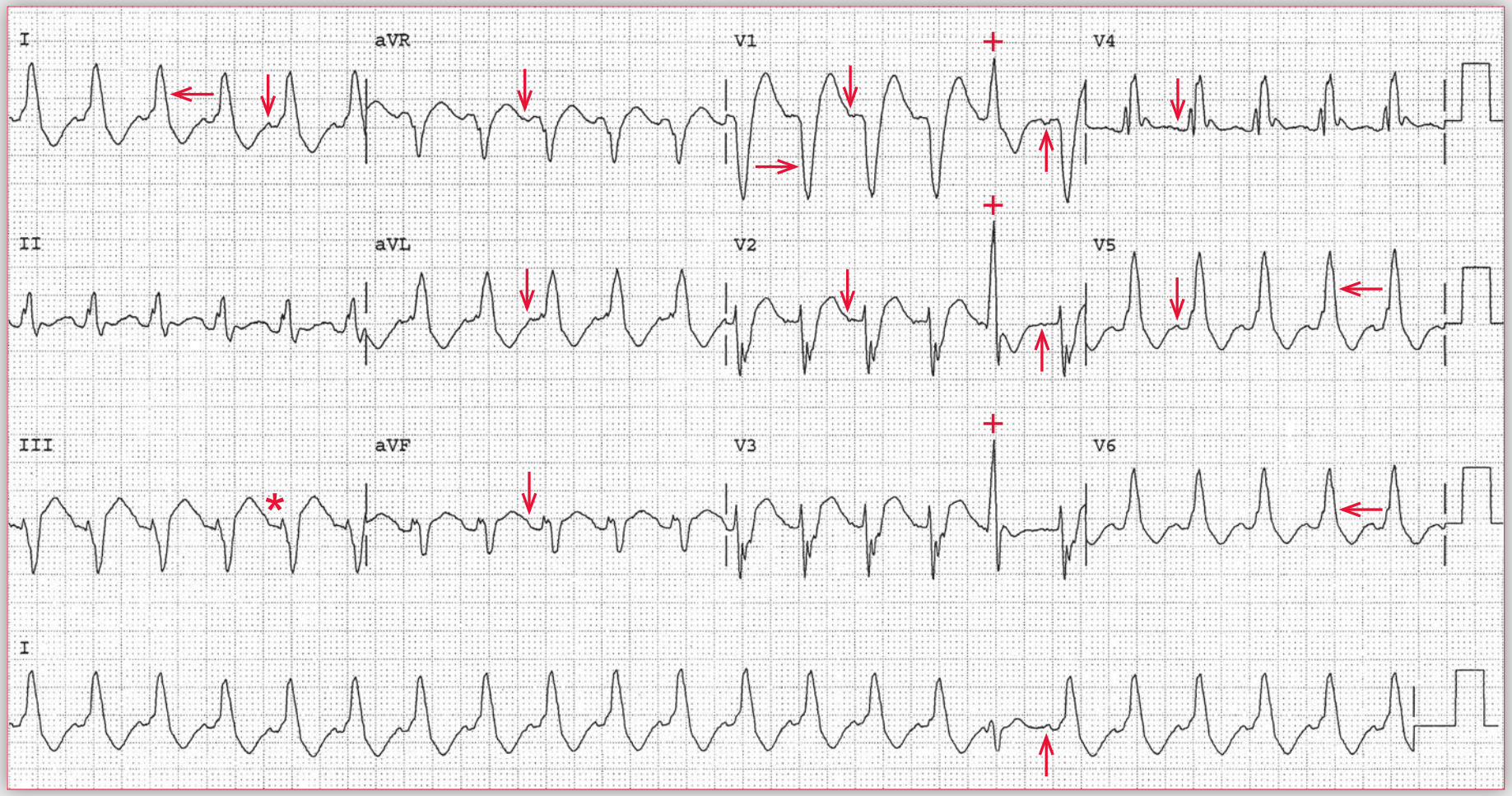
Notes

Practice Case 81

You are consulted about a patient who underwent surgical hip repair 3 hours ago, during which there was significant intraoperative bleeding. The primary medical team is concerned about ventricular tachycardia on a routine postoperative ECG.

What is the rhythm diagnosis?





ECG 81 Analysis: Sinus tachycardia, left bundle branch block, premature ventricular complex

There is a regular rhythm at a rate of 138 bpm. P waves are not obviously present, although notching (*) of the terminal part of the T wave can be seen in lead III. In the presence of tachycardia, P waves are often seen at the end of the T waves, especially if the PR interval is prolonged. T waves should have a smooth upstroke and downstroke; therefore, any notches or bumps on the T wave are strongly suggestive of superimposed P waves. In addition, P waves may become apparent during pauses. Noted is an isolated premature ventricular complex (+), after which there is a pause and a P wave (↑) can be seen. The PR interval is 0.14 second. Having established the baseline PR interval, it is clear that there are indeed P waves (↓) in front of each of the QRS complexes and that the notching of the T wave in lead III is the superimposed P wave. The P waves are positive in leads I, III, aVF, and V5-V6. Hence this is sinus tachycardia.

The QRS complex duration is prolonged (0.16 sec), and the morphology is that of a left bundle branch block (LBBB), with a broad R wave in leads I and V5-V6 (←) and QS complex morphology in lead V1 (→). The axis in the frontal plane is leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals (320/490 msec) are slightly prolonged. However, the prolonged QRS complex must be considered. If the QT interval is corrected for the prolonged QRS interval by reducing it by 0.06 second, then the QT/QTc intervals are normal (260/395 msec).

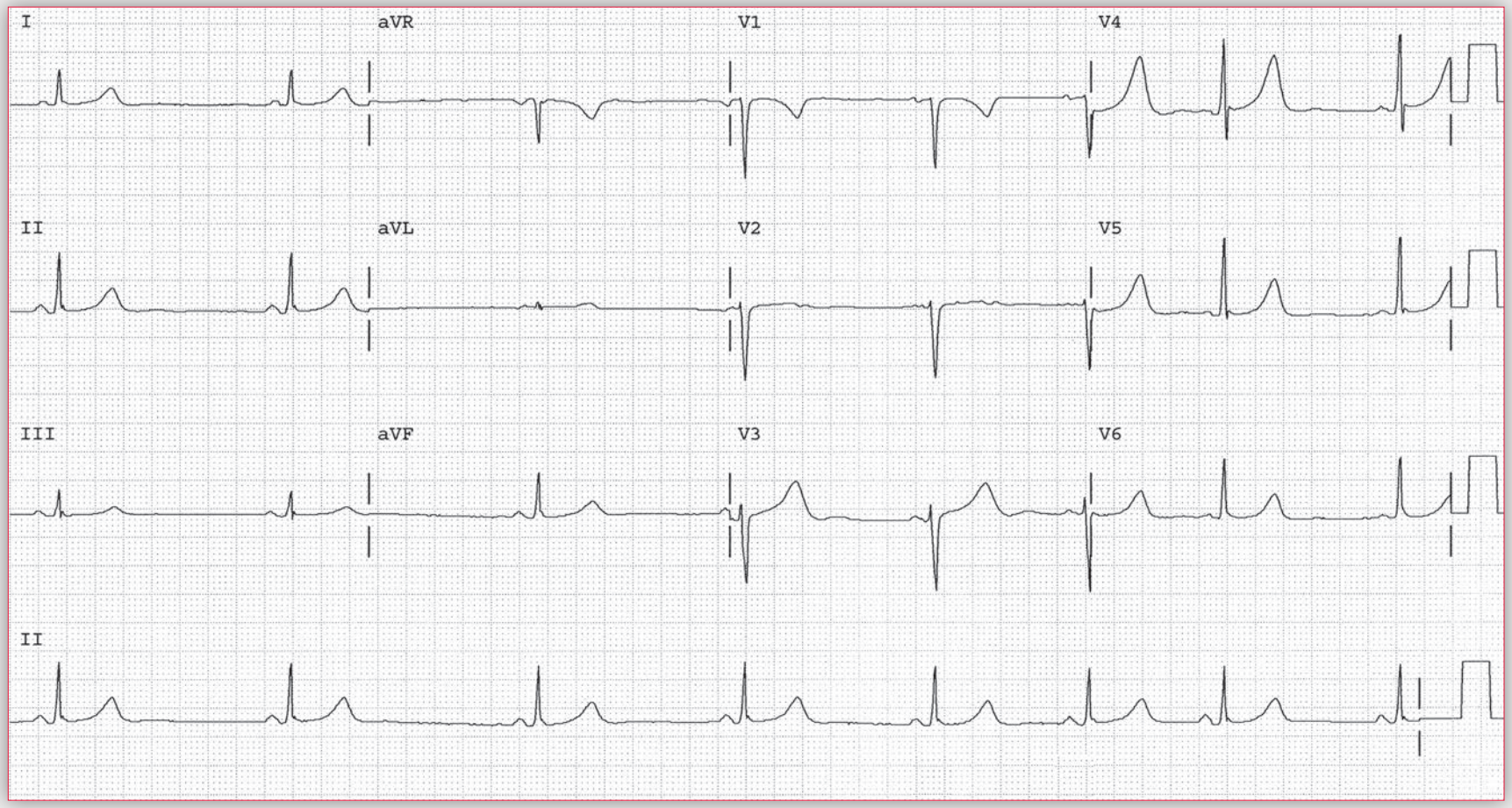
Hence, this is sinus tachycardia with aberrancy due to an underlying bundle branch block and not ventricular tachycardia. Sinus tachycardia in the postoperative setting commonly results from blood loss, dehydration, infection, or pulmonary embolism. The LBBB may be preexistent or rate related, but one would need to compare prior ECGs to make this determination. If the LBBB is a new finding, it may warrant further clinical evaluation (*ie*, echocardiogram, stress testing) when the patient is more stable to assess for structural heart disease or coronary disease. ■

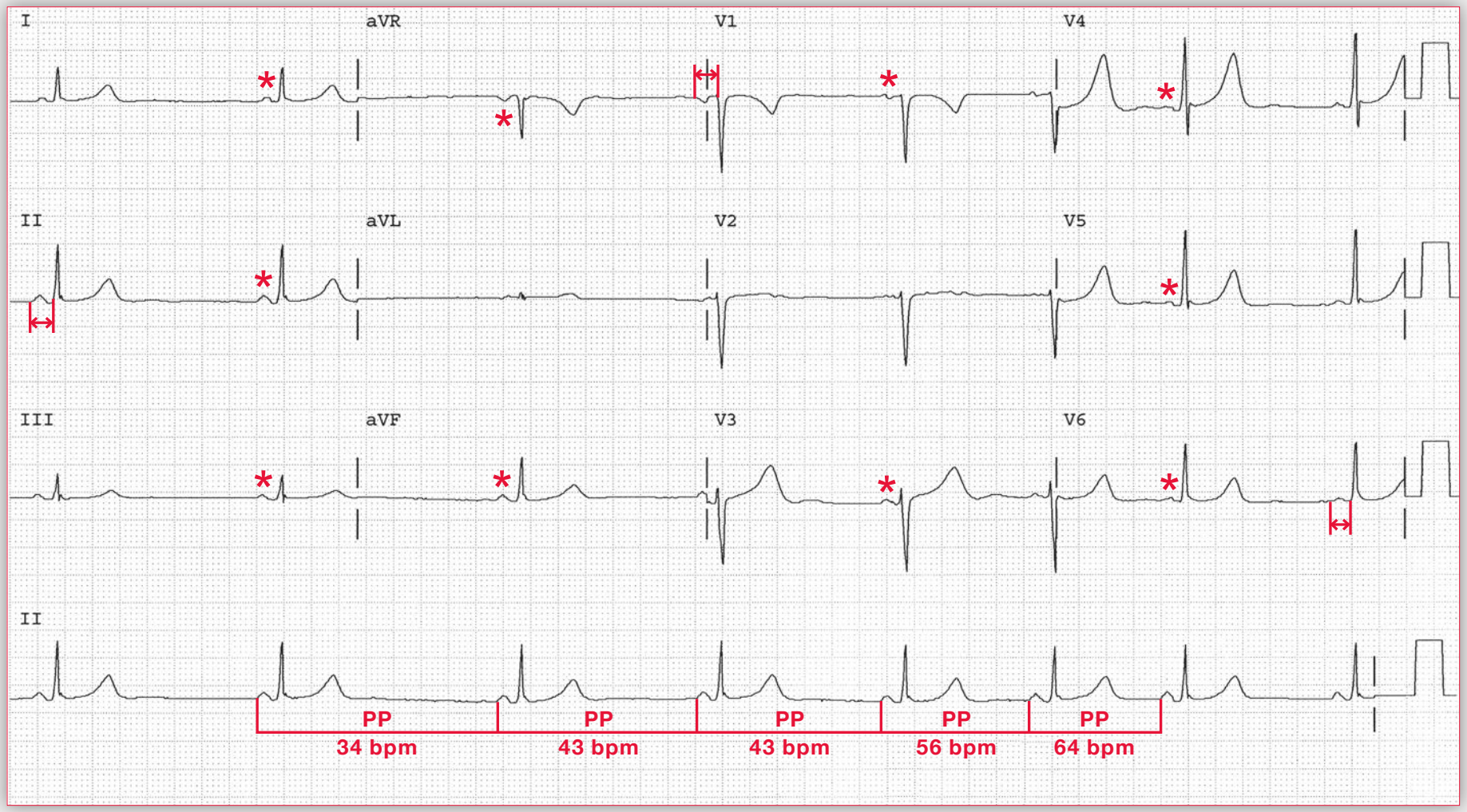
Notes

Practice Case 82

Which of the following individuals is most likely to be associated with the following ECG?

A. A marathon runner
B. A diabetic patient
C. A patient with primary amyloidosis
D. A patient with dehydration





ECG 82 Analysis: Sinus arrhythmia

The rhythm is irregularly irregular with a heart rate ranging between 34 and 64 bpm. The QRS complex duration (0.08 sec) and morphology are normal. The axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (500/450 msec). There are only three supraventricular rhythms that are irregularly irregular. These include sinus arrhythmia in which there is one P-wave morphology and a constant PR interval; multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm), in which there are three or more P-wave morphologies without any one being dominant; and atrial fibrillation in which there are no organized P waves. A P wave (*) with the same morphology is seen before each QRS complex, and the PR interval (↔) is constant (0.16 sec). This is, therefore, sinus arrhythmia, which is a respirophasic arrhythmia.

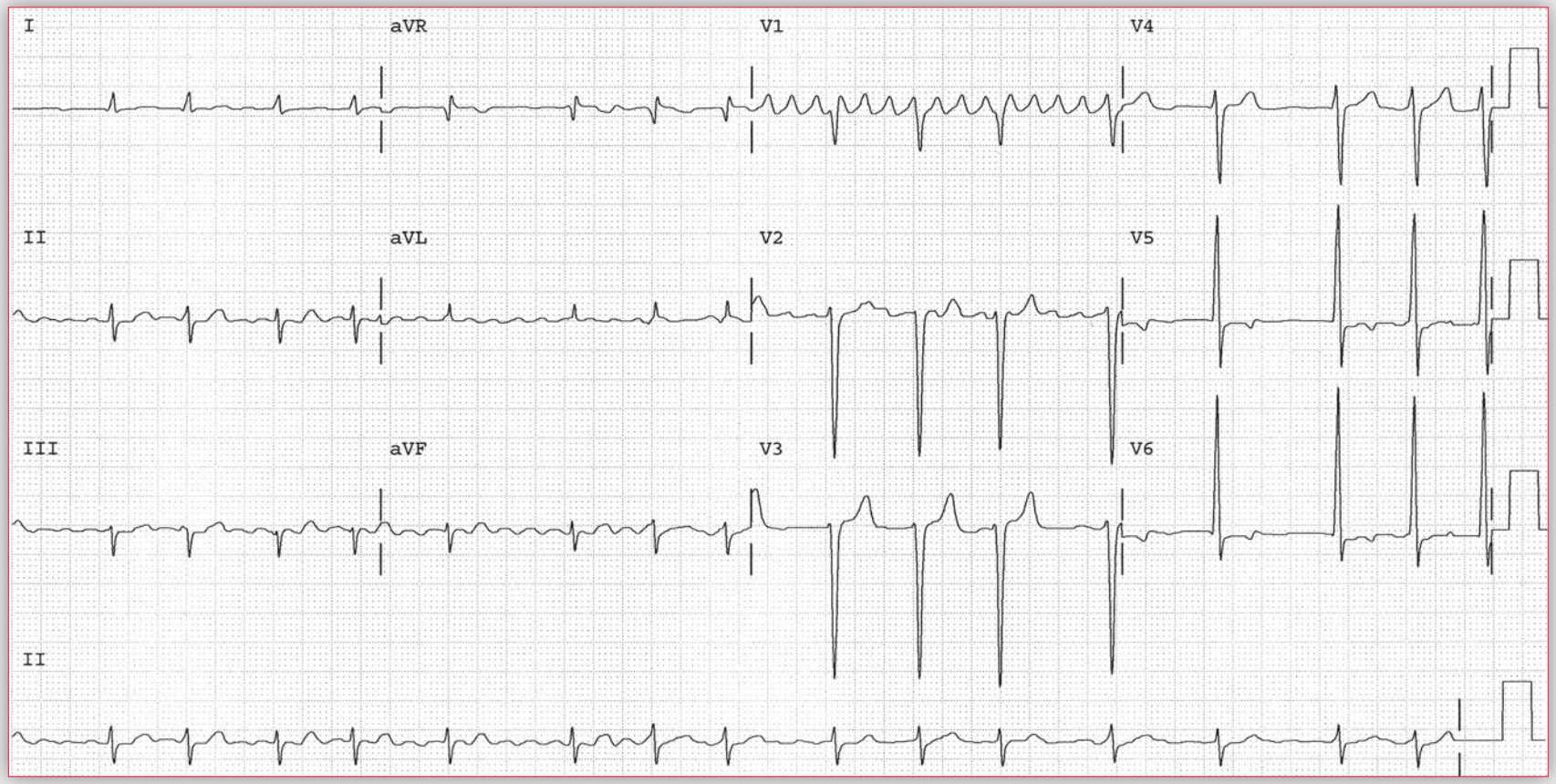
Sinus arrhythmia is a reflection of cardiac vagal tone because the vagus nerve mediates changes in heart rate due to changes in cardiac preload from respiration (Bainbridge reflex). With inspiration and increased preload resulting in atrial stretch, vagal tone is reduced, sinus node automaticity is enhanced, and heart rate is increased; with expiration, preload decreases, sinus node automaticity decreases, vagal tone increases, and heart rate slows. Healthy individuals such as marathon runners exhibit high degrees of cardiac vagal tone and would be most likely to have sinus arrhythmia and hence this ECG finding. Patients with autonomic dysfunction such as those with diabetes and amyloidosis exhibit less vagal tone than a normal individual. States of satiety activate parasympathetic stimulation of the vagus nerve and the respirophasic effect, whereas states of starvation or dehydration blunt this respirophasic effect because the sympathetic nervous system is activated in these states. ■

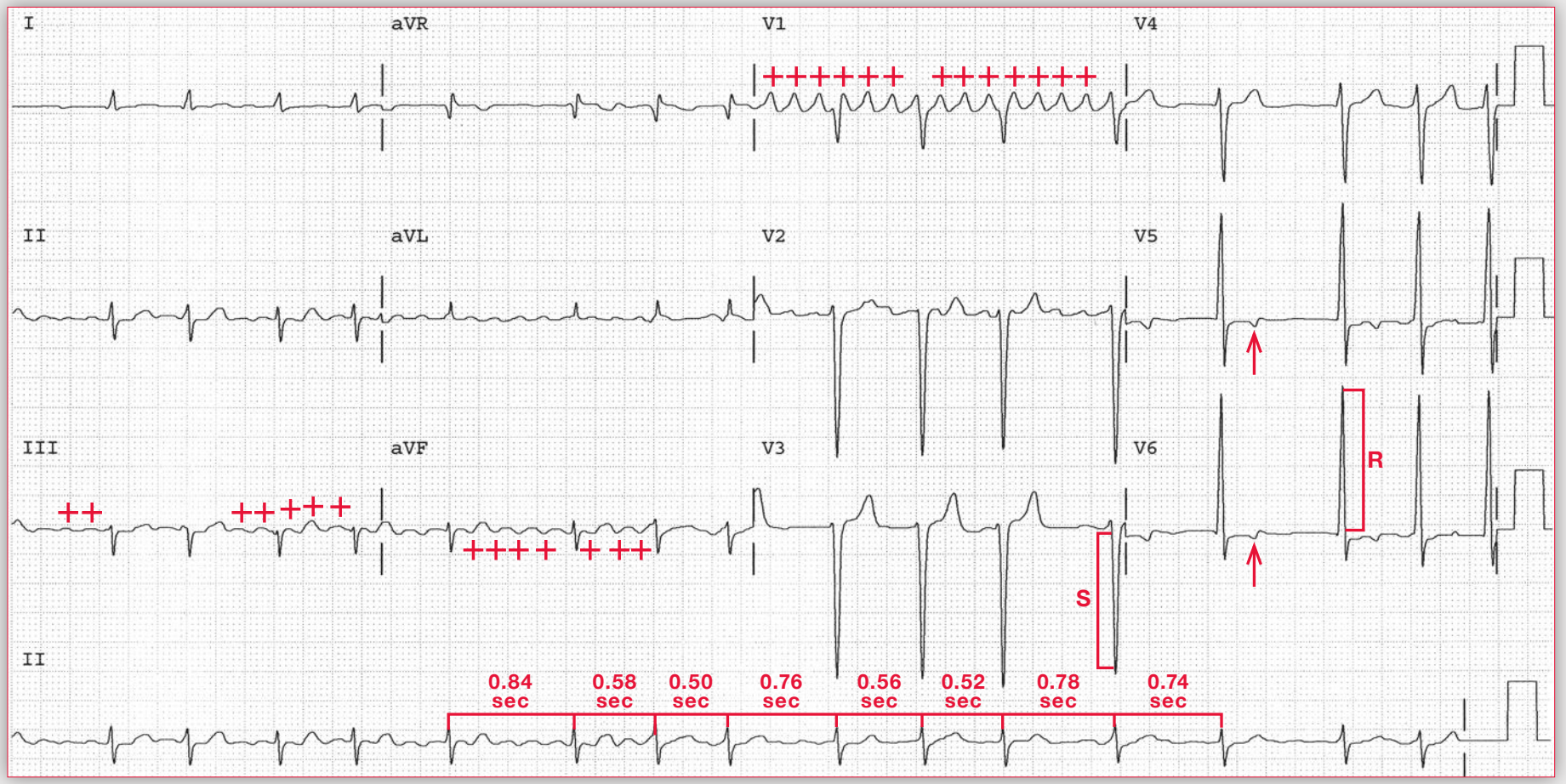
Notes

Practice Case 83

A 46-year-old patient with diabetes and hypertension presents to her primary physician's office with complaints of 1 week of limiting breathlessness with normal daily activities. She denies chest pressure and orthopnea. Her vital signs are remarkable only for a blood pressure of 158/82 mm Hg. On physical exam, the only notable finding is an irregular apical impulse. The physician is surprised not to have heard an S4 as she has record of hearing a loud S4 in the past. She promptly obtains an ECG.

What findings on the ECG explain the patient's symptoms and physical exam?





ECG 83 Analysis: Coarse atrial fibrillation, left ventricular hypertrophy, left axis, clockwise rotation (poor R-wave progression and late transition)

The rhythm is irregularly irregular without any organized P wave. The average rate is 104 bpm. There is evidence of atrial activity with coarse waveforms that are irregular in morphology, amplitude, and interval, best seen in leads II, III, aVF, and V1 (+). The atrial rate is higher than 320 bpm. This is, therefore, coarse atrial fibrillation. While the waveforms are prominent and resemble atrial flutter waves, particularly in lead V1, they demonstrate irregularity in morphology, amplitude, and interval (as seen in leads II, III, aVL, and aVF) and also have a rapid rate. In contrast, the waves in atrial flutter are uniform in morphology, amplitude, and interval, in contrast to what is seen on this ECG. In addition, the ventricular response rate with atrial flutter may be irregular but with a pattern (*ie*, regularly irregular). In contrast, the RR intervals are irregularly irregular in atrial fibrillation.

The QRS complex is of normal duration (0.08 sec) and the axis is leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (320/420 msec). The voltage in the limb leads is low (< 5 mm in each limb lead). However, the QRS complex amplitude is increased (S-wave depth in lead V3 = 25 mm [[ ] and R-wave amplitude in lead V6 = 23 mm [ ]]; S-wave depth in lead V3 + R-wave amplitude in lead V6 = 48 mm), diagnostic for left ventricular hypertrophy (*ie*, S-wave depth in lead V3 + R-wave amplitude in lead V5 ≥ 35 mm). There are T-wave abnormalities in leads V5-V6 (↑), which are probably secondary to left ventricular hypertrophy.

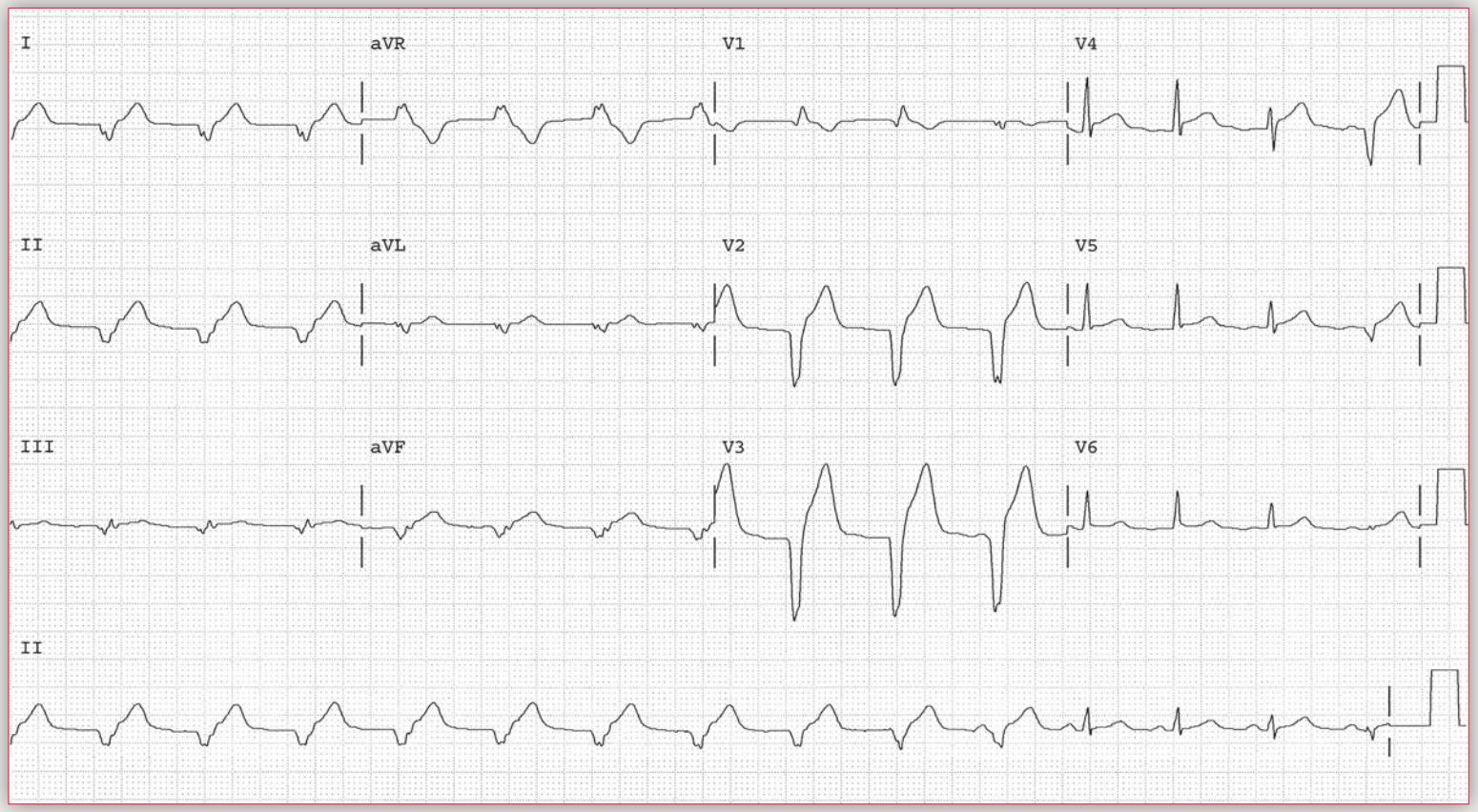
There is poor R-wave progression across the precordium, a result of clockwise rotation in the horizontal plane. In addition to the axis determined in the frontal plane, there is also an axis in the horizontal plane, determined by imagining the heart as viewed from under the diaphragm. Normally there is a gradual increase in the R-wave amplitude from leads V1-V6, with a normal QRS transition point (R/S > 1) occurring at leads V3-V4. Clockwise rotation is present when there is poor R-wave progression with late transition. R-wave amplitude does not increase normally and transition (R/S > 1) occurs at leads V4-V5. This is due to the fact that left ventricular forces develop late as a result of electrical rotation of the left ventricle posteriorly. Counterclockwise rotation is present when there is early transition (R/S > 1 in lead V2). This is due to the fact that the left ventricular electrical axis is rotated such that left ventricular forces develop early.

With the development of atrial fibrillation, which is likely recent in onset in view of the coarse fibrillatory waves (*ie*, > 2 mm in amplitude), there is a loss of atrial contraction. Therefore, S4 is absent as this represents atrial contraction producing rapid flow into a noncompliant left ventricle as is present with left ventricular hypertrophy. The symptoms are most likely the result of atrial fibrillation with the loss of atrial contraction, necessary to fill the noncompliant ventricle, as well as the rapid ventricular rate that often occurs with exertion as a result of sympathetic enhancement of AV conduction. Initial therapy would be better control of the ventricular rate, especially with exercise. More definitive therapy (*ie*, restoration of sinus rhythm vs. maintenance of atrial fibrillation with anticoagulation or antiplatelet therapy) would be based on several factors, including whether symptoms are controlled with adequate rate control. If not, restoration of sinus rhythm would be necessary to reestablish normal hemodynamics given the presence of diastolic dysfunction and a noncompliant ventricle. Also important would be a discussion with the patient to determine her preference. ■

Practice Case 84

A 67-year-old man presents to the emergency department with shortness of breath, palpitations, and chest discomfort. An ECG is obtained (ECG 84A). Several minutes later a second ECG (84B) is obtained. Based on the results of this second ECG, a procedure is performed.

ECG 84A



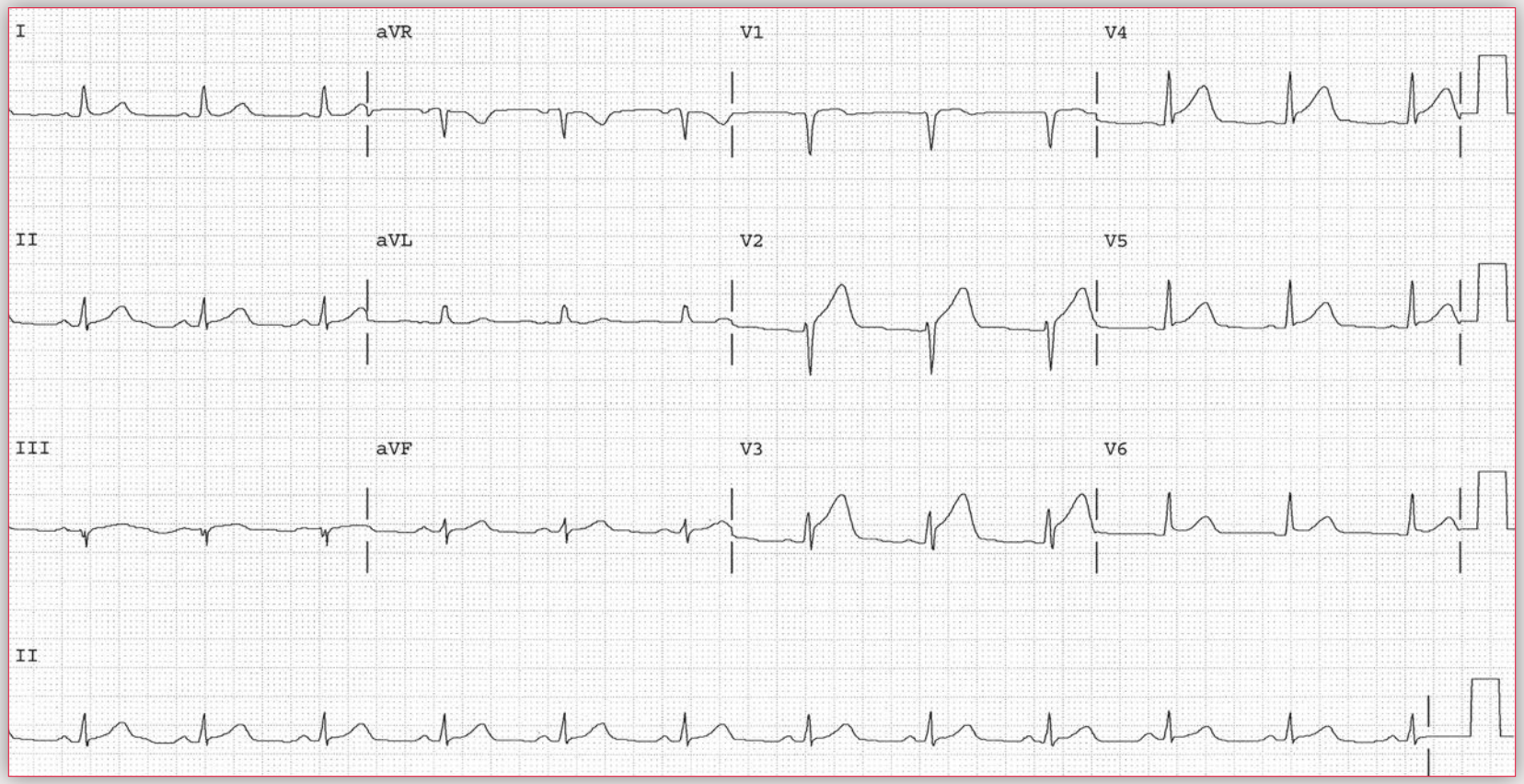
Practice Case 84

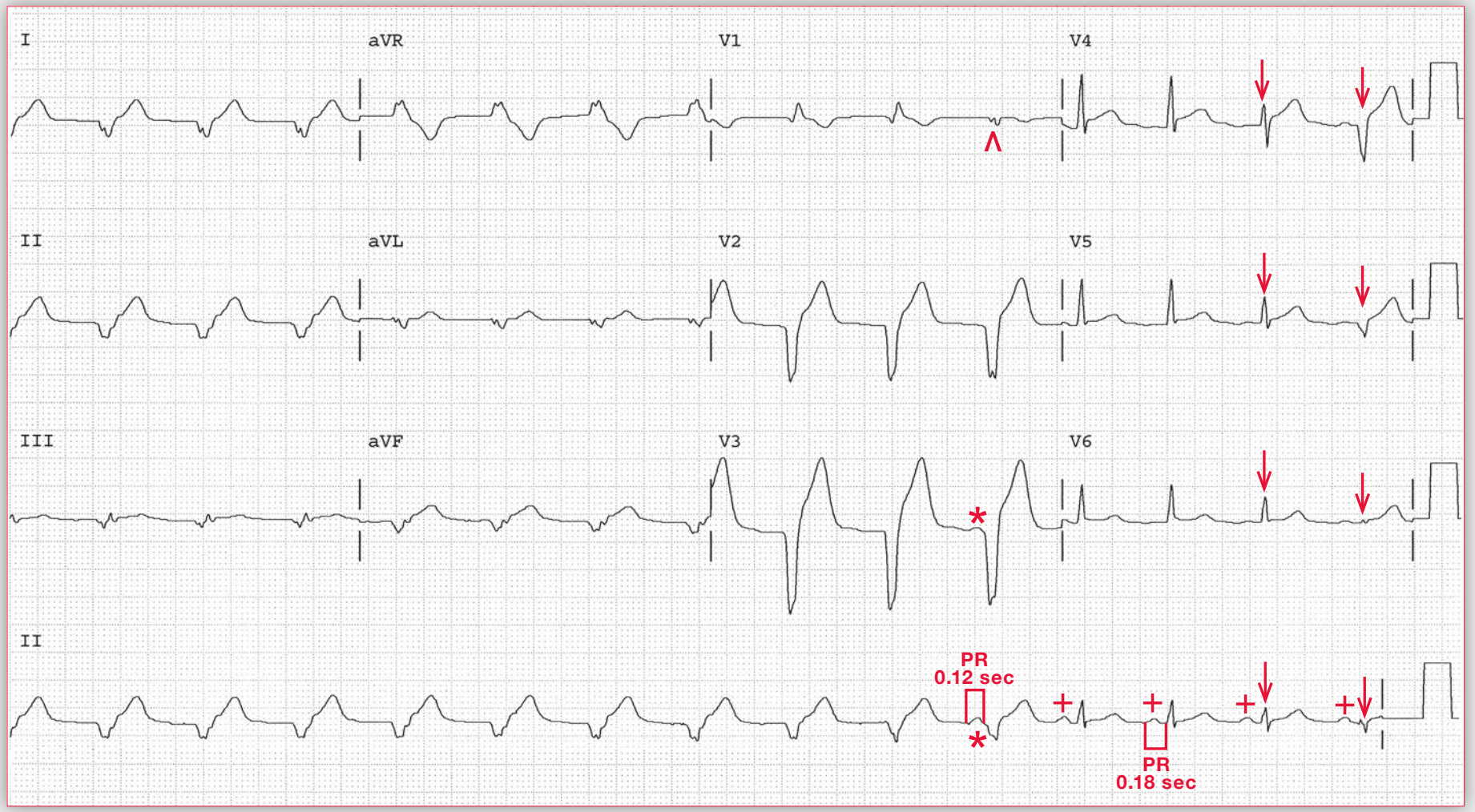
What is the etiology of the rhythm seen in ECG 84A?

After which of the following cardiac tests or procedures is the rhythm in ECG 84A most likely to be seen?

A. Treadmill exercise test
B. Tilt table test
C. Electrophysiology study
D. Percutaneous coronary intervention
E. Percutaneous aortic valvuloplasty

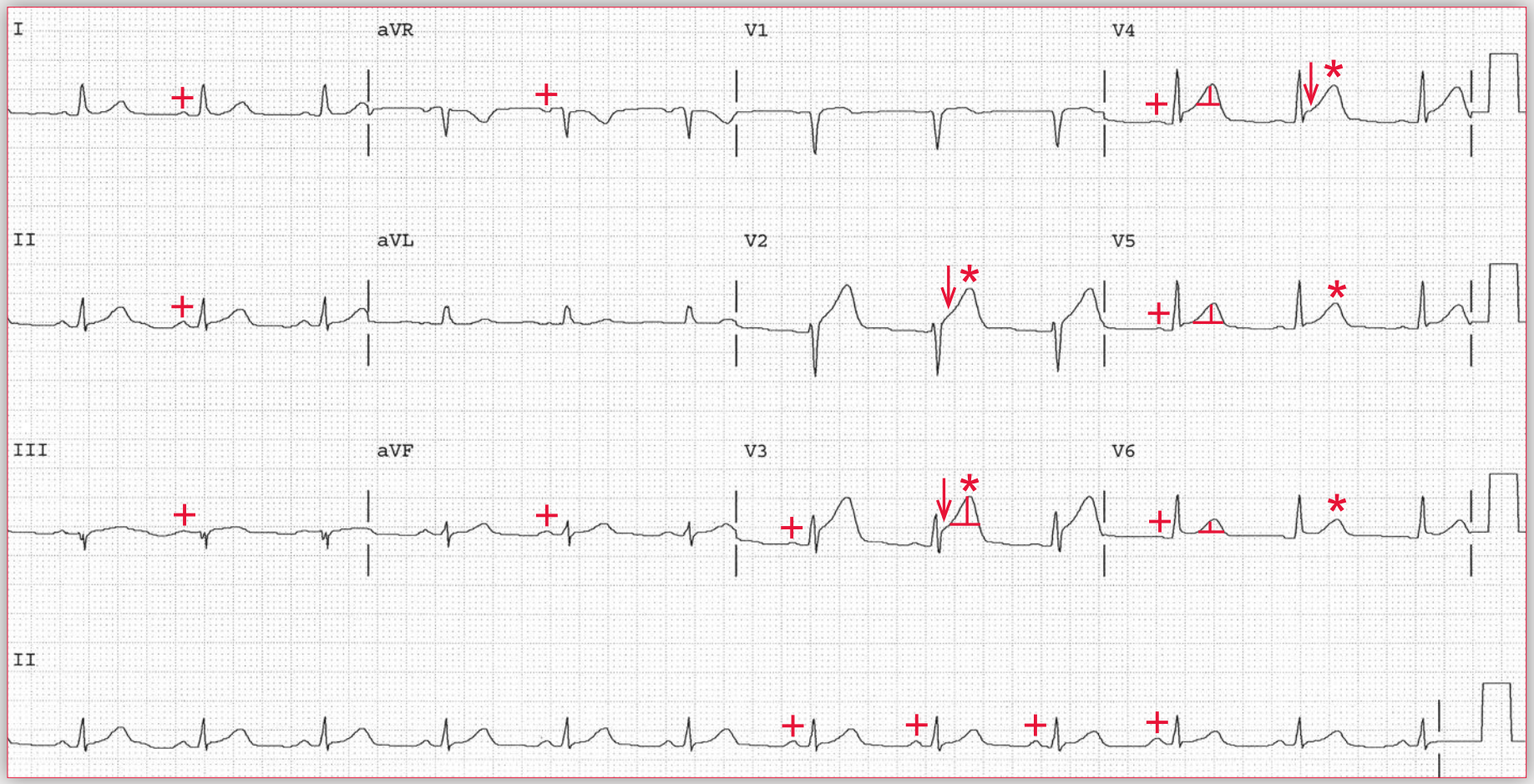
ECG 84B





ECG 84A Analysis: Accelerated idioventricular rhythm, normal sinus rhythm

In **ECG 84A** the rhythm is regular at a rate of 90 bpm. The QRS complex duration is increased (0.16 sec), the QRS morphology is abnormal (resembling neither a typical right nor left bundle branch block), and the axis is indeterminate, between –90° and +/-180° (negative QRS complex in leads I and aVF). No obvious P waves are seen during the initial portion of the ECG. However, a P wave (*) can be seen before the 10th QRS complex, which has a similar duration and morphology as those that precede it, although in lead V1 the QRS complex is different (^). The PR interval is short (0.12 sec). However, the 11th and 12th QRS complexes are narrow and there is a P wave (+) before each of these with a stable PR interval (0.18 sec). The P wave is upright in leads II and V4-V6. These are, therefore, sinus complexes. Hence the ECG shows an accelerated idioventricular rhythm (AIVR) with a fusion beat (^), after which sinus rhythm resumes. A fusion beat (which has a morphology that is different from either the ventricular or the sinus complex, although it resembles both) results from an impulse originating from the atria and conducting through the AV node–His-Purkinje system that fuses with an impulse that originates from the ventricular myocardium. The presence of fusion complexes or completely captured complexes (Dressler complex) during a wide complex rhythm means that there is AV dissociation and the rhythm is ventricular in origin. Also consistent with a ventricular origin for the rhythm is the presence of an indeterminate axis, which during a wide complex rhythm is seen only in situations where there is direct activation of the ventricular myocardium (ventricular complex, ventricular pacing [particularly biventricular pacing], or preexcitation due to Wolff-Parkinson-White pattern). Although the last two QRS complexes are preceded by P waves, their morphology is slightly different (particularly in leads V4-V5), suggesting that these are also fusion beats (↓) due to a recurrence of the AIVR related to either a slight slowing of the sinus rate or a slight increase in the rate of the AIVR, allowing it to be seen again.



ECG 84B Analysis: Normal sinus rhythm, acute anterior wall ST-segment elevation myocardial infarction

In **ECG 84B**, there is a regular rhythm at a rate of 70 bpm. There is a P wave (+) before each QRS complex with a constant PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm. The QRS complex duration is normal (0.08 sec) and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/430 msec). There is ST-segment elevation (↓) in leads V2-V4, and the T waves are symmetric (*). This is thus an acute anterior wall myocardial infarction. The marked difference in the QRS complexes between the tracings confirms that **ECG 84A** showed an AIVR.

An AIVR is often seen as a reperfusion arrhythmia after an acute myocardial infarction resulting from restoration of blood flow due to spontaneous lysis of thrombus, administration of a thrombolytic agent, or a percutaneous coronary intervention. It is usually transient and generally does not require any therapy. However, if associated with symptoms, it can be suppressed by a standard antiarrhythmic agent. It first needs to be established whether the ventricular rhythm is accelerated, in which case an antiarrhythmic drug can be given, or is an escape rhythm due to complete heart block, in which case a pacemaker should be inserted before suppressing the ventricular rhythm (if this is clinically indicated). If the atrial rate is faster than the ventricular rate, then this is complete heart block and an accelerated ventricular rhythm. If the atrial rate is slower than the ventricular rate, then this is AIVR. As the second ECG showed evidence of an acute ST-segment elevation myocardial infarction, the patient underwent urgent catheterization and a percutaneous coronary intervention was performed. ■

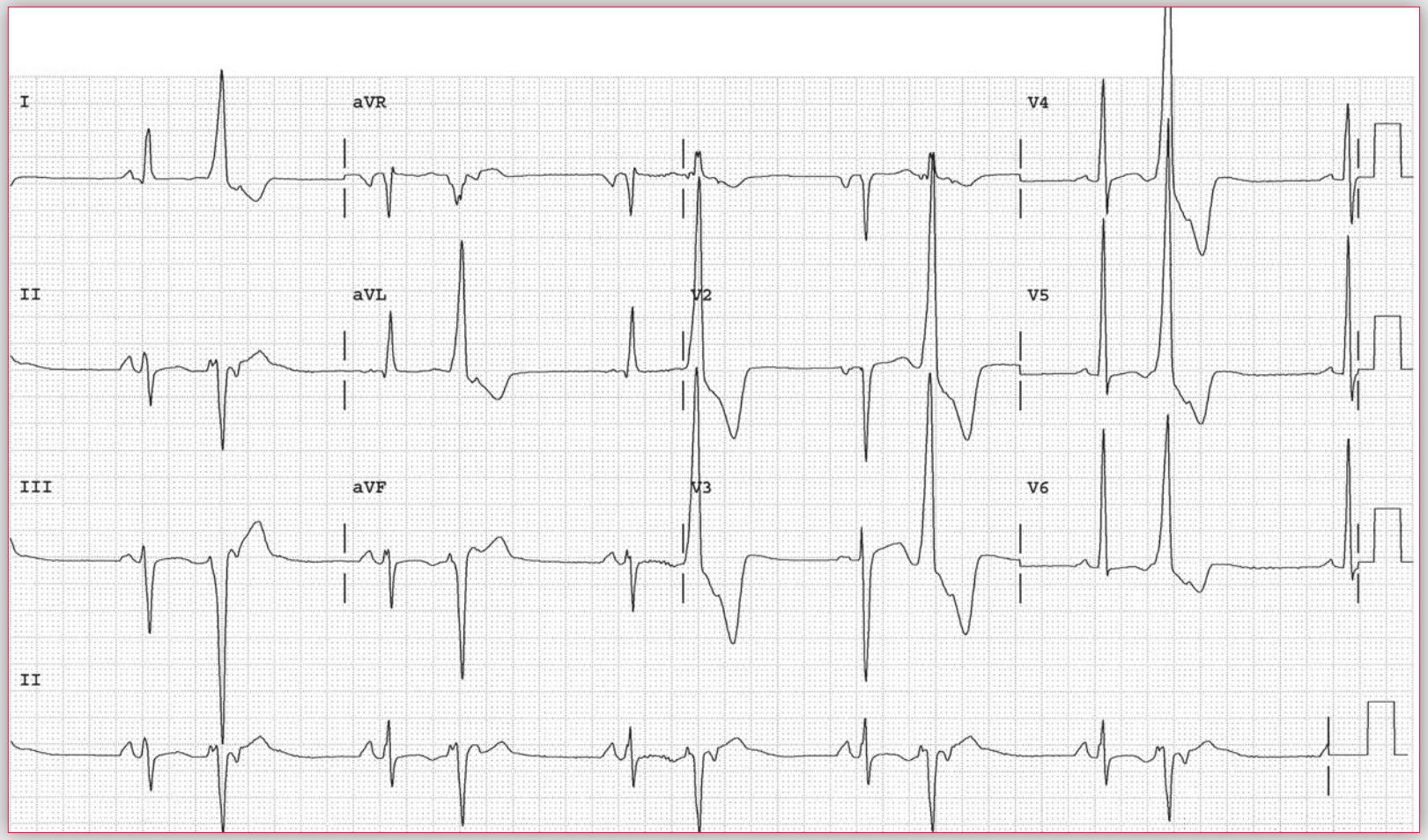
Notes

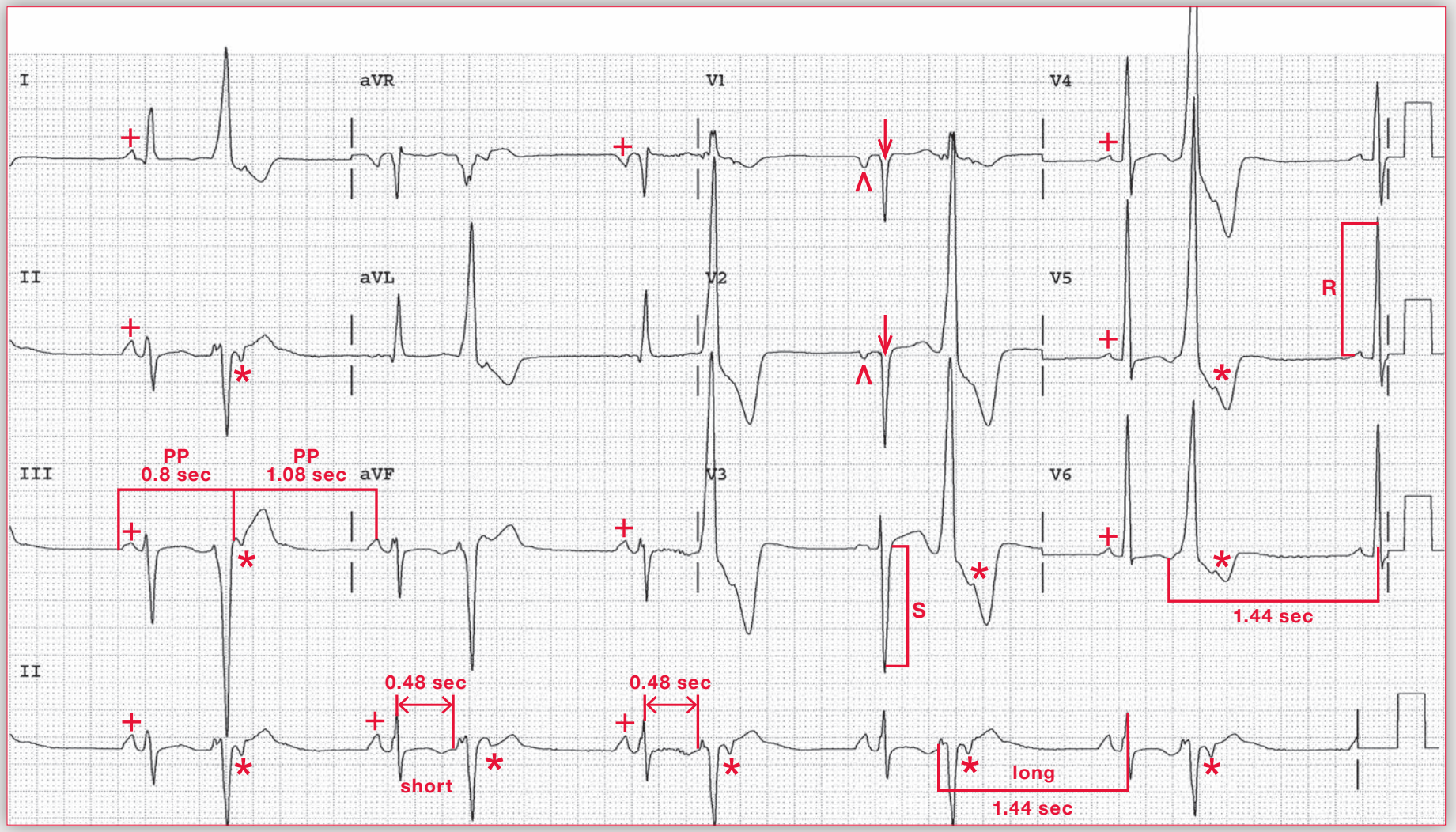
Practice Case 85

A 68-year-old man is being evaluated in the emergency department because of complaints of abdominal bloating and nausea. His pulse is felt to be irregular and as a result he is placed on telemetry. During an exam performed several minutes later his nurse notes that his heart rate, measured by manual pulse, is about half as much as is being calculated by the telemetry monitor.

What is noted on the ECG?

Why are the measured pulse and telemetered pulse different?





ECG 85 Analysis: Sinus rhythm, premature ventricular complexes in a bigeminal pattern (ventricular bigeminy), old anteroseptal myocardial infarction, left ventricular hypertrophy, left atrial hypertrophy (or abnormality)

The rhythm is regularly irregular with a repeating pattern of long (⊔) and short (↔) RR intervals. All the long intervals (1.44 sec) are the same, and the short intervals are the same (0.48 sec). The narrower QRS complexes (duration, 0.10 sec) are preceded by a P wave (+), and the PR interval is constant (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. The P wave is negative in leads V1-V2 (^), suggesting the presence of left atrial hypertrophy.

The axis is extremely leftward (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology); hence this is a left anterior fascicular block. The other etiology for an extreme left axis is an old inferior wall myocardial infarction in which there is a deep initial Q wave in leads II and aVF. There is a Q wave in leads V1-V2 (↓), suggesting an old anteroseptal myocardial infarction. In addition, the R-wave amplitude is increased in lead V5 (28 mm) and there is an increased S-wave depth in lead V3 (22 mm) ( ]); the R-wave amplitude in lead V5 + the S-wave depth in lead V3 = 50 mm ([ ), which is diagnostic for left ventricular hypertrophy (*ie*, S-wave depth in lead V3 + R-wave amplitude in lead V5 ≥ 35 mm).

After each sinus complex there is a wide (0.18 sec), abnormal QRS complex that does not have a preceding P wave. Its morphology is not typical for either a right or left bundle branch block. These are premature ventricular complexes (PVCs) and they are occurring in a bigeminal pattern (every other QRS complex is a PVC). The coupling interval (↔) between the sinus complex and the PVC is the same (*ie*, there is a fixed coupling interval), suggesting that there is a relationship between the two waveforms. As there is a fixed coupling interval, the PVCs are the result of a reentrant mechanism, activated by each sinus QRS complex and resulting in the impulse circulating around the circuit once.

Immediately following each premature complex in leads II and III is a distinct negative waveform (*). This waveform is also seen in leads V1-V6 and is within the ST segment (*). This is a P wave. It is different than the sinus P wave and is early with a shorter PP interval (0.8 sec) (⊔) compared with the interval to the next sinus P wave (1.08 sec) (⊓). Hence it is a retrograde P wave, resulting from the PVC and conducted retrogradely through the AV node to activate the atria. It is not an on-time sinus P wave as it is early and has a different morphology. ■

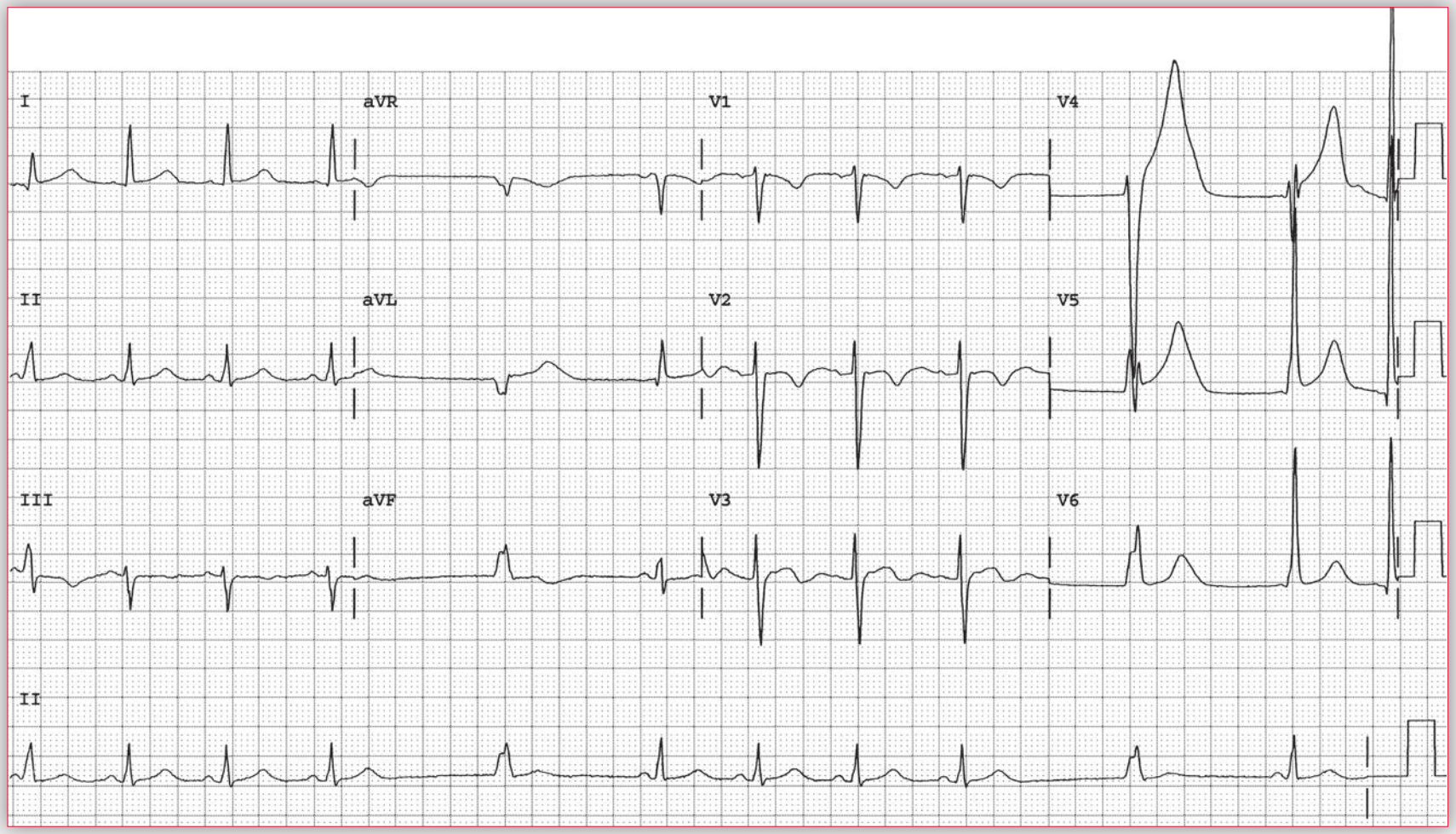
Notes

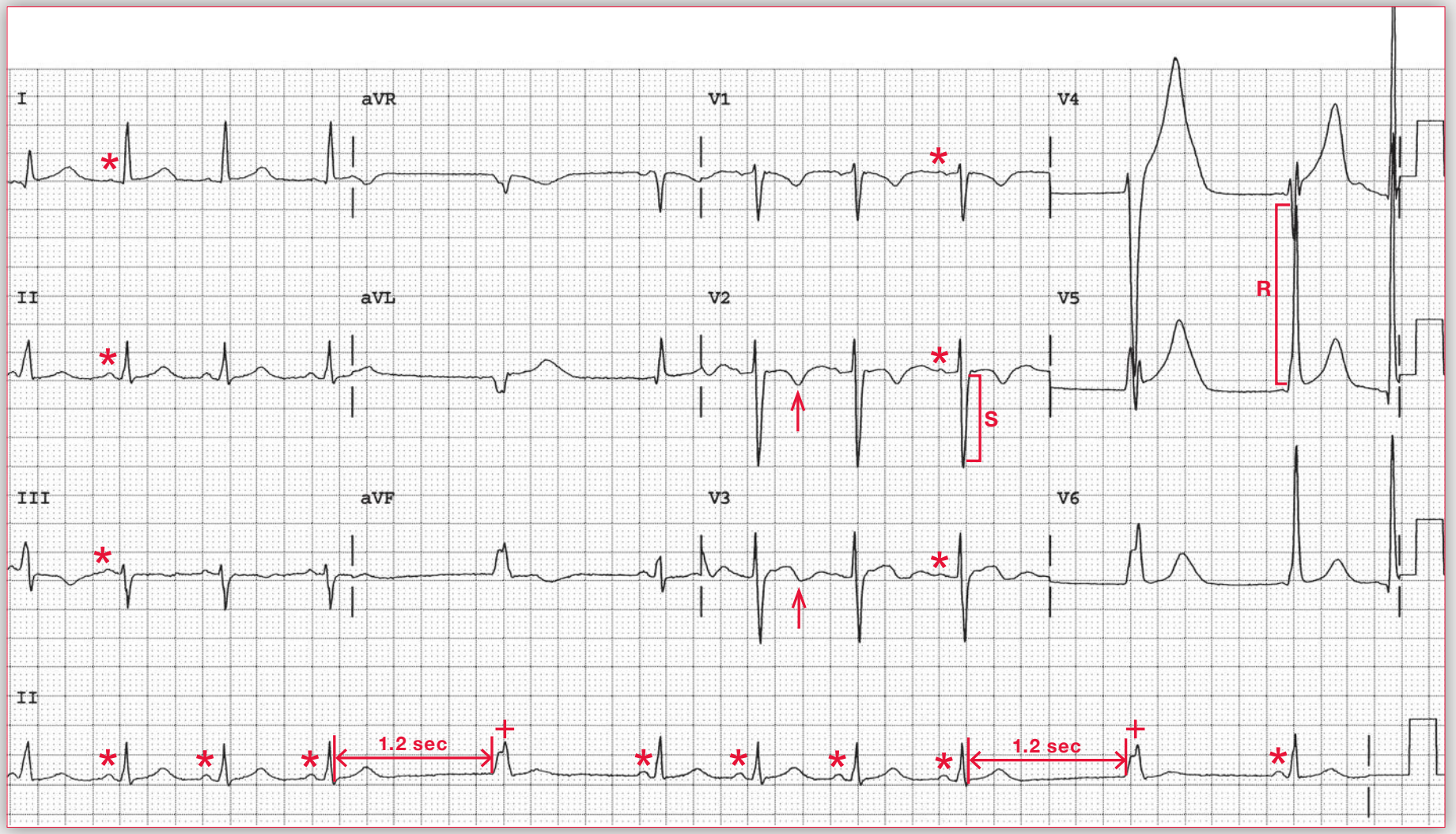
Practice Case 86

A 68-year-old, otherwise healthy man describes intermittent sensations of a "skipped heart beat" but denies palpitations, dizziness, lightheadedness, or syncope. You obtain the following ECG.

How would you describe the arrhythmia?

What are your next steps in management?





ECG 86 Analysis: Sinus node pause with ventricular escape complex, left ventricular hypertrophy with associated ST-T wave abnormalities

QRS complexes two through four and six through nine and the last QRS complex are preceded by a P wave (*), and the PR interval is constant (0.14 sec). These QRS complexes occur at a regular rhythm at a rate of 86 bpm, and the P waves are positive in leads I, II, aVF, and V5-V6. Hence there is an underlying sinus rhythm. The QRS complexes have a normal duration (0.08 sec) and morphology, but there is high voltage consistent with left ventricular hypertrophy (S-wave depth in lead V2 + R-wave amplitude in lead V5 = 65 mm). This meets one of the criteria for left ventricular hypertrophy (*ie*, S-wave depth in lead V2 + R-wave amplitude in lead V5 ≥ 35 mm). There are also nonspecific ST-T wave changes (↑) in leads V2-V3. The QT/QTc intervals are slightly prolonged (400/460 msec).

There are two long RR intervals of the same duration of 1.2 sec (↔), during which there is no evidence of atrial activity. These long intervals are the result of sinus node pauses as an on-time sinus P wave is not present. It is not clear whether this is due to sinus node arrest or exit block. As a result of the pause there is an escape QRS complex (+) that occurs at a rate of 50 bpm. The etiology of the escape complex (*ie*, junctional or ventricular) is based on its morphology and is not related to the escape rate. As the escape QRS complexes are wide with a morphology that differs from that of the sinus complexes, these are ventricular in origin.

A sinus pause may be due to either sinus node exit block or sinus node arrest. With sinus node exit block, the PP interval surrounding the pause is equal to two sinus PP intervals, while with a sinus node arrest the PP interval surrounding the pause is unrelated to the sinus rate and may be shorter or longer than two PP intervals. In this case the etiology is not clear, but it is most likely the result of a sinus node arrest. This is usually the result of a drug that affects the sinus node, such as digoxin, a β-blocker, or a calcium-channel blocker. It may also be the result of increased vagal tone or underlying sinus node dysfunction. The fact that the pause is ended by an escape ventricular complex suggests that there is either depression of an atrial or junctional focus or an acceleration of a ventricular focus.

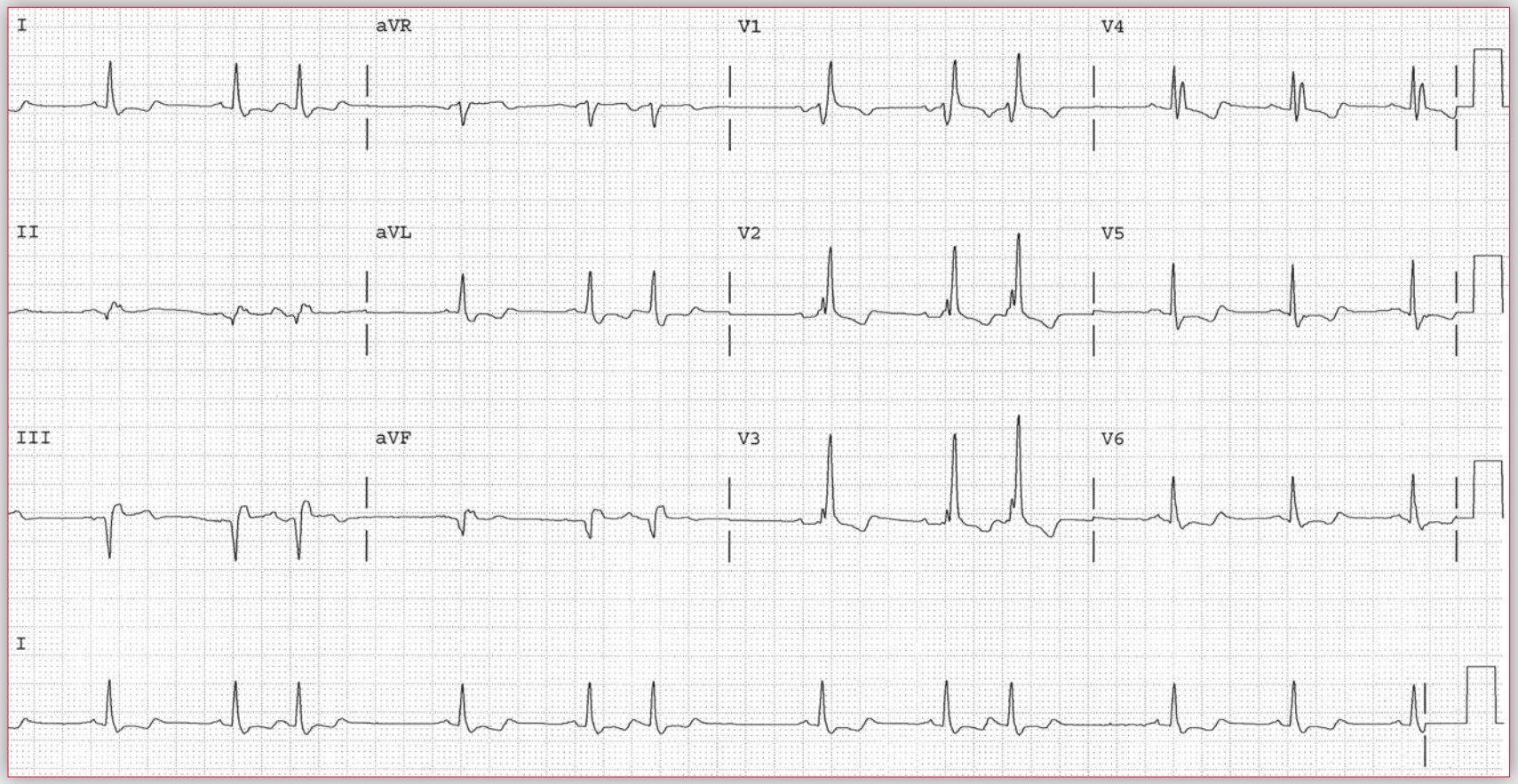
The patient has evidence of mildly symptomatic sinus node dysfunction. Symptoms are not a result of any hemodynamic abnormalities. At this point, placement of a pacemaker is not absolutely indicated. Often, however, patients can underestimate their true degree of symptoms or decrease their activity level to compensate for the presence of symptoms without realizing it. The next step would be to accurately assess the extent and severity of the patient's sinus node dysfunction (*ie*, Holter monitor or extended telephonic monitoring with documentation of any symptoms) and determine whether there is any bradyarrhythmia-induced limitation to exercise capacity (treadmill exercise tolerance test). If the patient's sinus node dysfunction is found to correlate with more significant symptoms, then this is a class I indication for placement of a permanent pacemaker. ■

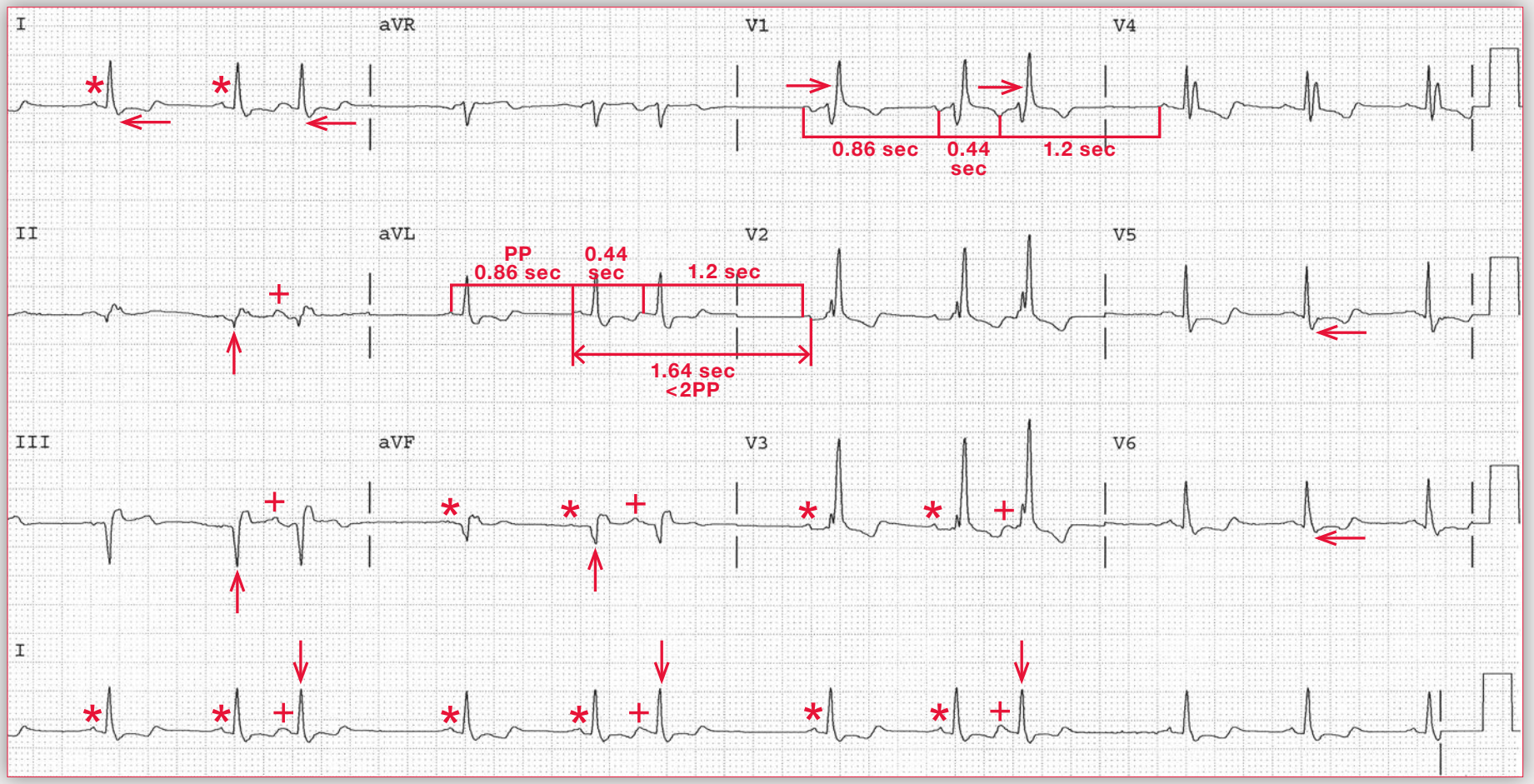
Notes

Practice Case 87

A 74-year-old man with coronary artery disease, prior myocardial infarction, and left ventricular dysfunction (ejection fraction 30%) presents with palpitations and has the following ECG.

How would you classify the arrhythmia?





ECG 87 Analysis: Normal sinus rhythm with atrial trigeminy, right bundle branch block, old inferior wall myocardial infarction, left axis, diffuse ST-T wave abnormalities

The rhythm is irregular, but there is a pattern to this irregularity with the appearance of group beating; hence the rhythm is regularly irregular. Every third complex (↓) is early and hence there is a trigeminal pattern. A P wave (*) of uniform morphology can be seen before the two complexes that are regular. The P wave is positive in leads I, II, aVF, and V4-V6. The PR interval is constant (0.16 sec), and the PP interval or rate (70 bpm) of these two complexes is identical each time. These are, therefore, two sinus complexes. The third QRS complex, which is premature (↓), also has a P wave (+) in front of it, but the P wave is of a different morphology. This premature complex has a fixed relationship with the preceding QRS complex (*ie*, PP interval = 0.44 sec). Hence these are premature atrial complexes in a trigeminal pattern (atrial trigeminy). The premature atrial complex has a pause after it, but the PP interval around the premature beat (1.64 sec) is less than two sinus PP intervals (0.86 × 2 = 1.72 sec); hence this is less than a full compensatory pause.

All of the QRS complexes have the same morphology. The QRS complex duration is increased (0.12 sec), and there is a right bundle branch block morphology (RSR′ morphology in lead V1 [→] and a broad S wave in leads I and V5-V6 [←]). The axis is physiologically leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). However, the negative QRS complexes in lead aVF are the result of Q waves (↑); along with the Q wave in lead III, this indicates that a prior inferior wall myocardial infarction is the cause for the left axis. The QT/QTc intervals are 400/430 msec and 380/400 msec when corrected for the prolonged QRS duration.

Similar to atrial bigeminy, atrial trigeminy indicates that there is a repeating pattern of premature complexes. This is a benign arrhythmia with no important clinical implications except that there are frequent premature atrial complexes. No therapy is necessary unless the premature complexes are associated with symptoms or if the complexes are a "trigger" for a sustained atrial arrhythmia. This patient complains of palpitations. This symptom is usually the result of post-extrasystolic potentiation. The premature complex is followed by a pause during which the left ventricle continues to fill with blood, resulting in an increase in end-diastolic volume. The increased volume produces an increase in stroke volume during contraction as a result of the Frank-Starling effect and an increase in left ventricular inotropy. While therapy with an anti-arrhythmic agent may suppress the premature atrial complexes, eliminating the palpitations, an alternative therapy is the use of a β-blocker, which may result in a decrease in left ventricular inotropy and a reduction in the sensation of palpitations. ■

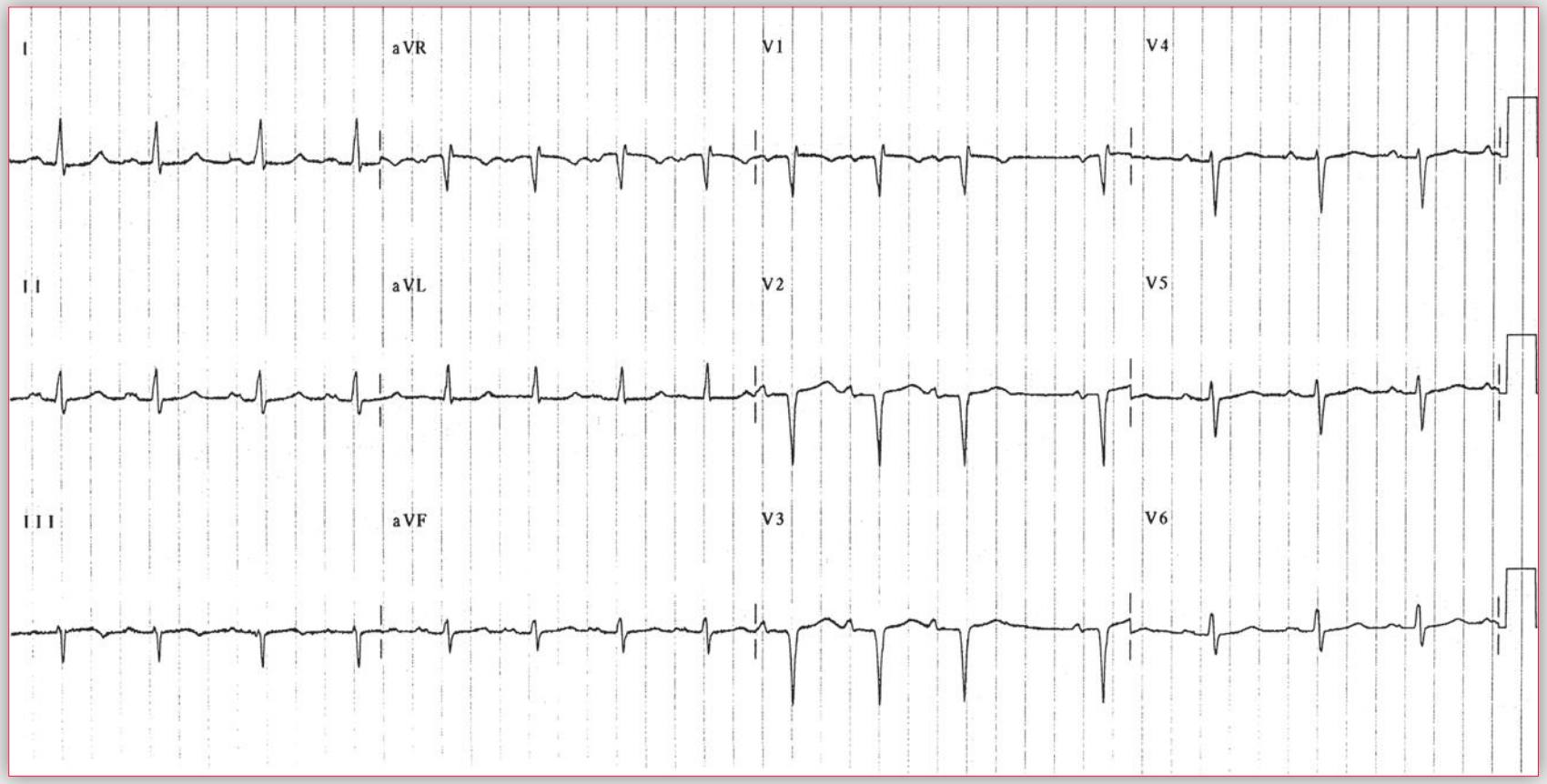
Notes

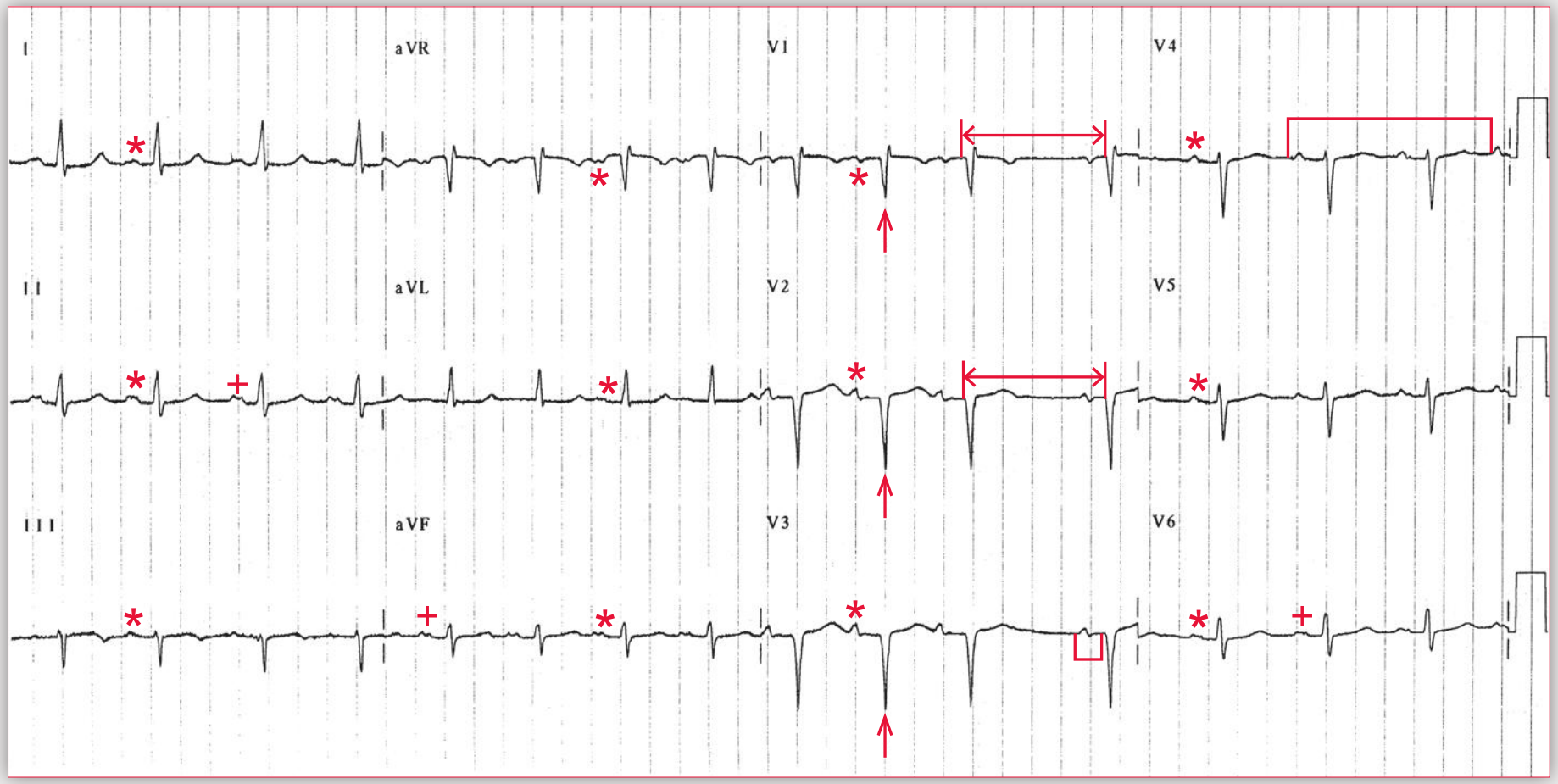
Practice Case 88

An 85-year-old woman with prior myocardial infarction (MI) and paroxysmal atrial fibrillation treated with metoprolol and warfarin comes for a routine visit. Physical examination reveals clear lung fields, a normal jugular venous pressure, and a normal apical impulse. A prominent S1 with a mid-diastolic murmur preceded by a snap is heard on auscultation at the apex. A routine ECG is obtained.

What are the ECG abnormalities?

What is the clinical diagnosis?





ECG 88 Analysis: Sinus node arrest, P mitrale or left atrial hypertrophy, prior anteroseptal MI

There is a regular rhythm at a rate of 96 bpm. There is a P wave (*) before each QRS complex, and the PR interval is constant (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm. The P wave in leads II, aVF, and V6 is broad (>120 msec) and prominently notched (+); this is termed P mitrale and indicates left atrial hypertrophy. The QRS complex duration is normal (0.08 sec), and there is a normal axis of approximately 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). The QT/QTc intervals are normal (320/404 msec).

There is one prolonged RR (or PP) interval of 1 second (↔); no P wave is seen during this interval, which rules out AV block or premature atrial complex as a cause for the pause. This is thus a sinus pause. There are two mechanisms for a sinus pause. The first is sinus node exit block. In this situation, the sinus node generates an impulse on time, but there is intermittent failure of the impulse to exit from the sinus node area into the atrium; hence the atrium is not activated. Therefore, the PP interval surrounding the pause is identical to two sinus intervals. The second mechanism is sinus node arrest. In this situation the sinus node fails to generate an impulse. Hence the duration of the pause is unrelated to the underlying sinus interval, and it may be longer or shorter than two sinus intervals. In this patient, the duration of the pause is less than two sinus intervals (⊓); therefore, this is a sinus node arrest. The PR interval of the complex after the pause (⊔) is slightly shorter (0.16 sec) than the baseline PR interval. The shorter PR interval may be due to faster AV conduction as a result of the very slow rate and more time for the AV node to recover. However, it is also possible that the QRS complex may be a junctional escape complex rather than a conducted sinus complex. In addition, leads V1-V3 have Q waves (↑), indicating a previous anteroseptal myocardial infarction.

Mitral stenosis is most commonly due to rheumatic heart disease. Other less common causes include congenital mitral valve abnormalities or mitral annular calcification. A left atrial myxoma may present with signs and symptoms similar to mitral stenosis. Increased resistance to diastolic flow from the left atrium to the left ventricle results in a mid-diastolic murmur heard best at the apex that may be preceded by a snap, which is known as the opening snap of the mitral valve (reflecting the opening of the stenotic mitral valve). S1 becomes more prominent, reflecting the increased intensity of closure (snapping closed) of a stiffened mitral valve with the two leaflets that are closer together and not widely opened at end-diastole due to the stenosis. The classic clinical findings of mitral stenosis include left atrial hypertrophy with elevated left atrial pressures; this progresses to the development of pulmonary hypertension and right ventricular failure. In some patients with elevated right-sided pressures, sinus node dysfunction may occur. This patient has evidence of left atrial hypertrophy on the ECG, which also increases the risk for atrial arrhythmias such as atrial fibrillation.

Atrial tachyarrhythmias can be particularly symptomatic in patients with mitral stenosis who depend on a sufficient diastolic filling time and atrial contraction to maintain normal left ventricular filling and stroke volume. Hence, it is essential to control heart rate adequately in patients with mitral stenosis and atrial fibrillation using either β-blockers, calcium-channel blockers, or digoxin. Restoration of sinus rhythm is often necessary to maintain adequate hemodynamics. ■

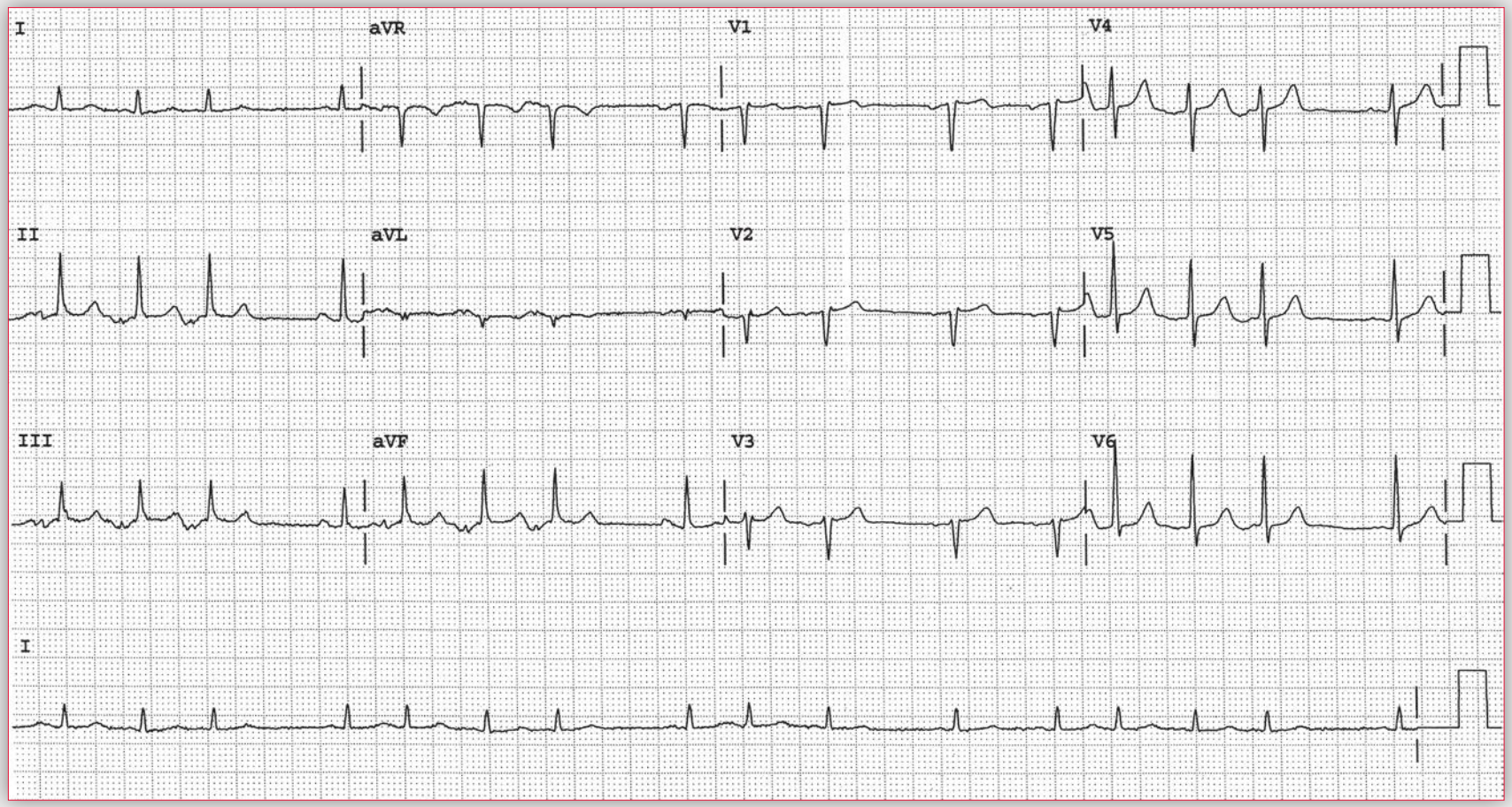
Notes

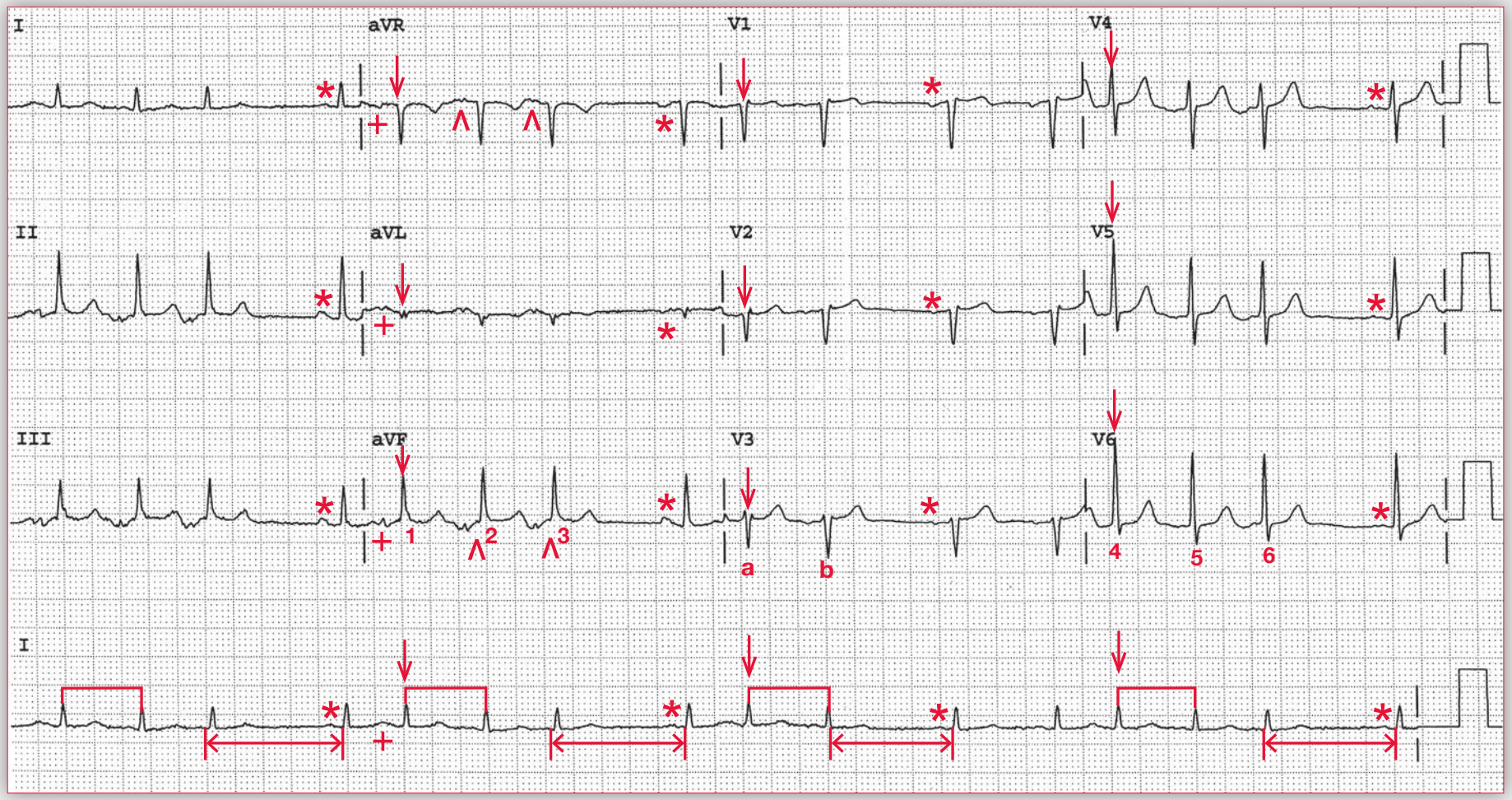
Practice Case 89

A 21-year-old woman arrives in the emergency department with palpitations. She has been up late studying for her college exams and drinking many cups of coffee. She notes that she feels a fluttering in her chest and feels anxious. The emergency department physician notes that her heart sounds are regularly irregular. She appears tired and mildly tremulous.

What does her ECG show?

What advice can you give this patient about her concerns that she may have a serious cardiac disorder?





ECG 89 Analysis: Atrial triplets and couplets

The rhythm is irregular, although there is a pattern that can be seen, as the long intervals are identical (↔) and there is regularity to many other RR intervals (⊓). Hence the rhythm is regularly irregular and the average rate is 96 bpm. Noted after each long RR interval is a P wave (*) that is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus P wave and the PR interval associated with each of these P waves is the same (0.18 sec). Therefore, this is the baseline PR interval. After the sinus complex there is an early or premature QRS complex (↓) that is preceded by a P wave. As seen in lead aVF, the premature P wave (+) has a different morphology compared with the sinus P wave. This is a premature atrial complex (PAC). Following this complex are two additional atrial complexes that have the same abnormal P wave (^). The rates of these three atrial complexes are 150, 100, and 130 bpm. Three sequential atrial complexes (1,2,3) is called an atrial triplet. In lead V3 there are two sequential PACs (a, b), which is termed an atrial couplet, and there is another atrial triplet (4,5,6) that follows two sinus beats.

All the QRS complexes have an identical duration (0.08 sec) and morphology, both of which are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/350 msec).

PACs are benign and rarely indicate a serious cardiac pathology. They are seen commonly in patients with normal hearts. They may be provoked by various factors, including medications (*eg*, digoxin or sympathomimetic agents such as theophylline), chemicals (caffeine or nicotine), or various illnesses (*eg*, chronic lung disease, chronic renal failure). These factors may also increase the frequency of PACs and can result in the occurrence of repetitive forms. PACs are also seen in patients with structural heart disease such as mitral valve disease (mitral valve prolapse or mitral regurgitation), hypertension, or cardiomyopathy and heart failure of any etiology. Hence this patient should be reassured that she does not have any serious cardiac problem. She should be advised to reduce her coffee intake and get more sleep. ■

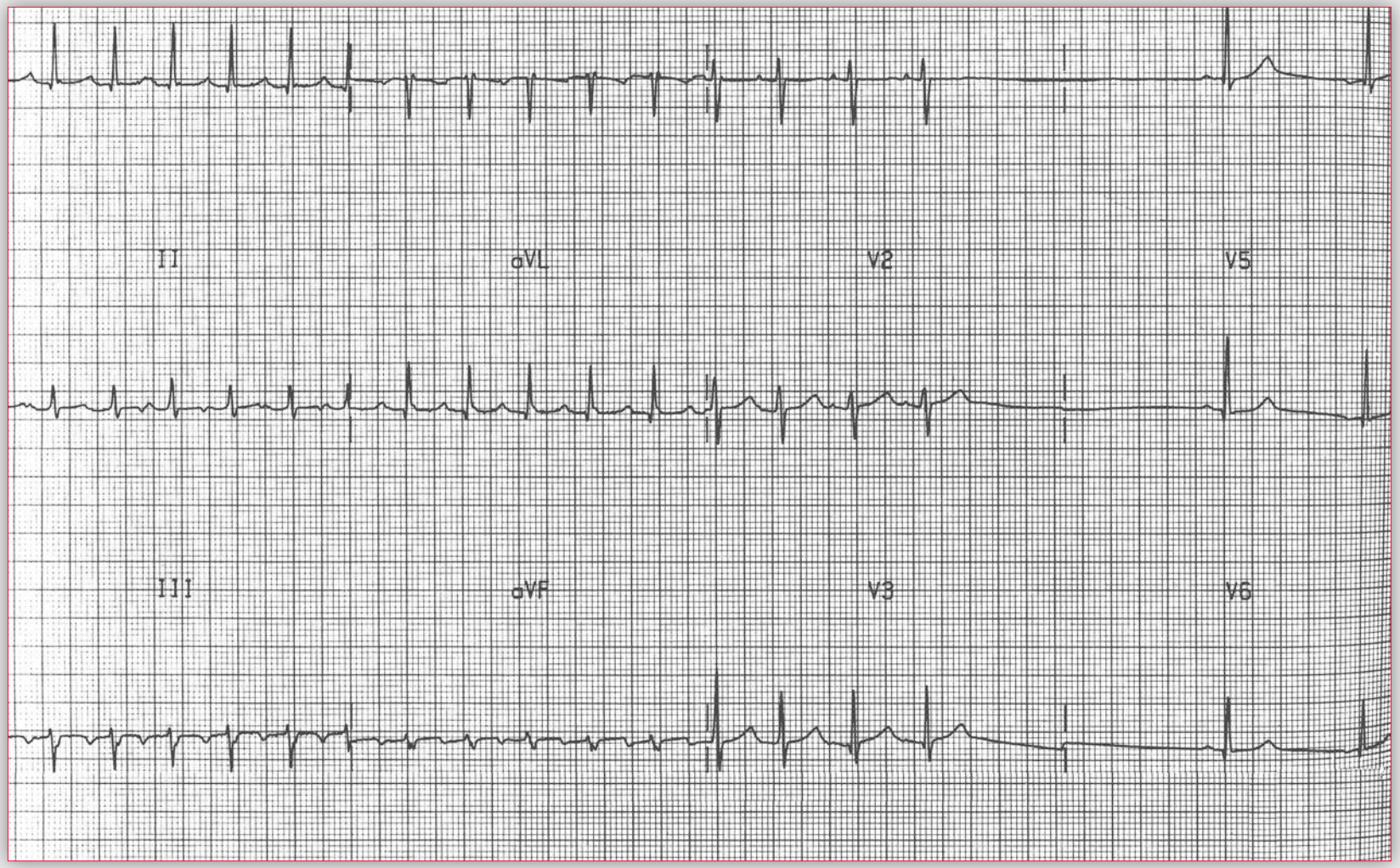
Notes

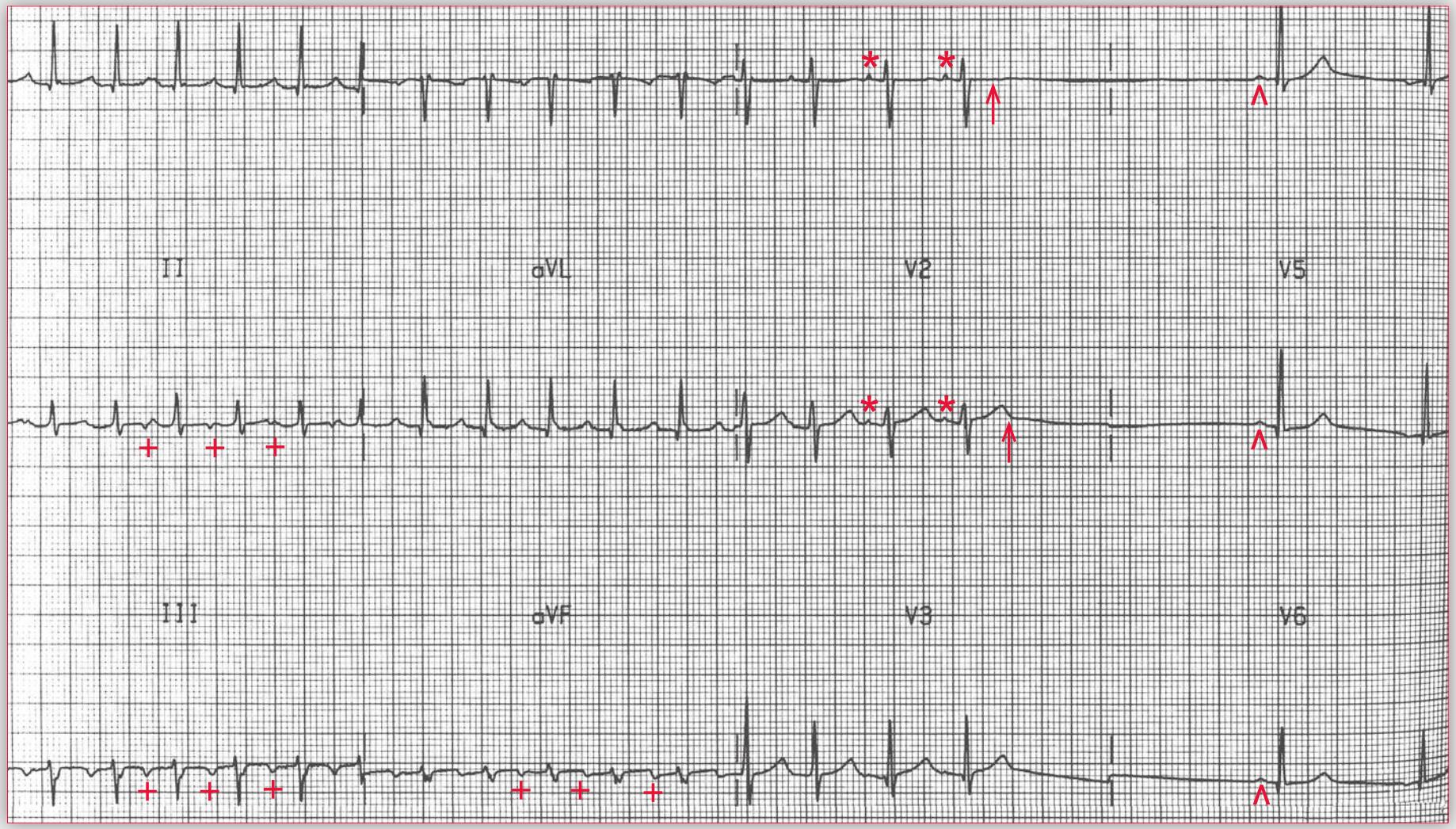
Practice Case 90

A 53-year-old woman presents to the emergency department with palpitations. The sensation came on suddenly and has lasted for about 40 minutes. While in the emergency department, an ECG is obtained and the palpitations stop abruptly.

What is the mechanism of the arrhythmia?

What key clues are present on the ECG to establish this as the etiology?





ECG 90 Analysis: Atrial tachycardia with termination to sinus rhythm

The initial part of the ECG shows a regular rhythm at a rate of 140 bpm. Negative (inverted) P waves (+) can be seen in leads II, III, and aVF, while P waves can also be seen in leads V1-V2 (*). The P waves are negative in leads II and aVF. The PR interval is stable at 0.12 second. The RP interval is 0.36 second. Hence this is a long RP tachycardia. Etiologies for a long RP tachycardia include sinus tachycardia with a first-degree AV block (not the case here as the P waves are negative in leads II and aVF), ectopic junctional tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block (not likely as a second atrial waveform is not seen), atypical atrioventricular nodal reentrant tachycardia (*ie*, fast-slow), or atrioventricular reentrant tachycardia. The tachycardia slows to a rate of about 130 bpm before abruptly terminating to a regular rhythm that is at a slower rate (60 bpm) and has a P wave (^) with a PR interval of 0.16 second. This is, therefore, a sinus complex. It should be observed that the tachycardia terminates without a P wave (↑) (*ie*, there is no P wave after the last QRS complex). This is the manner in which atrial arrhythmias terminate as the atrial focus stops firing. Hence this is atrial tachycardia. Further support for atrial tachycardia as the etiology is the gradual slowing of the rate prior to termination. This is often seen with arrhythmias that are due to an ectopic focus, which may manifest changes in automaticity and hence in rate.

The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). The QT/QTc intervals are normal (320/470 msec). ■

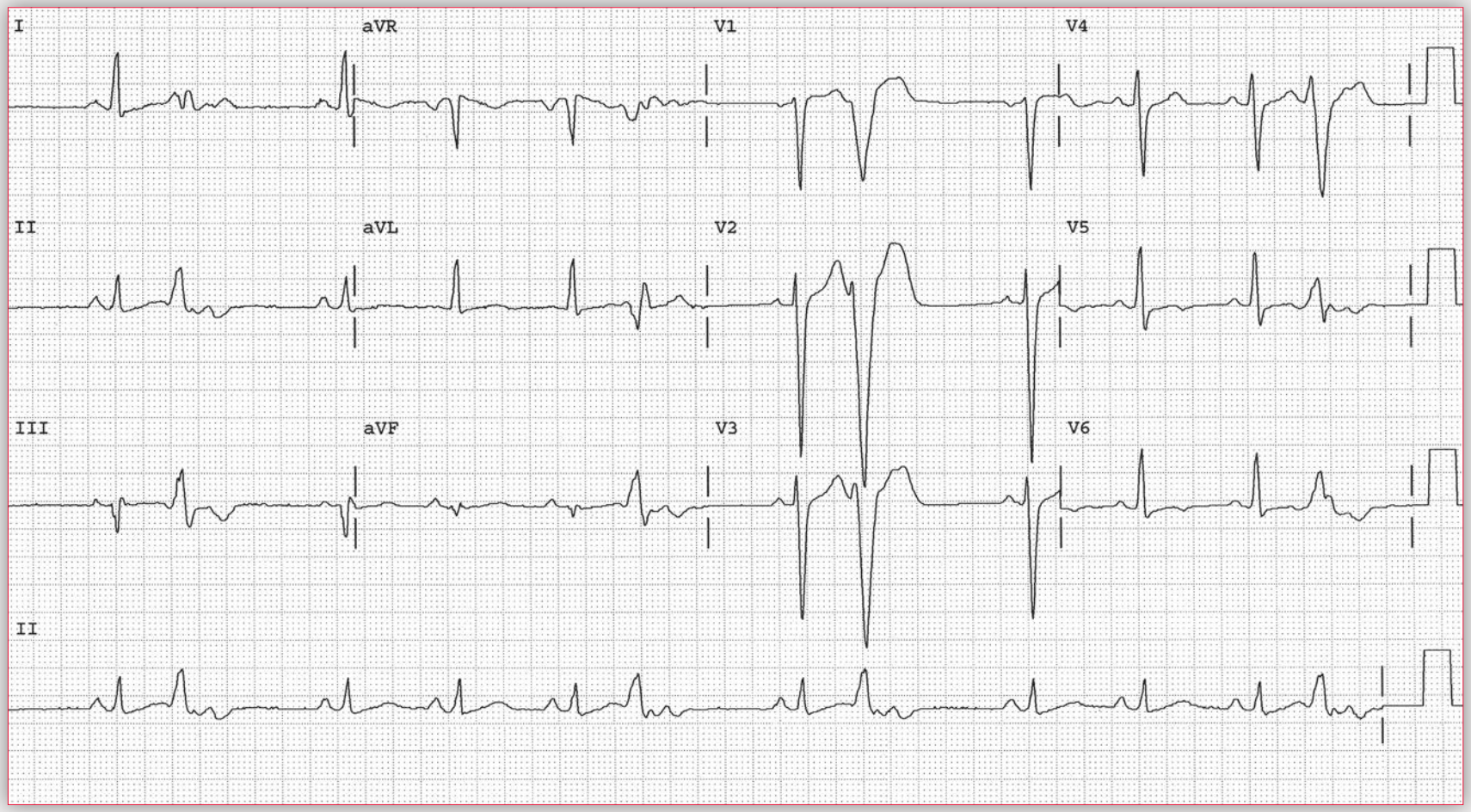
Notes

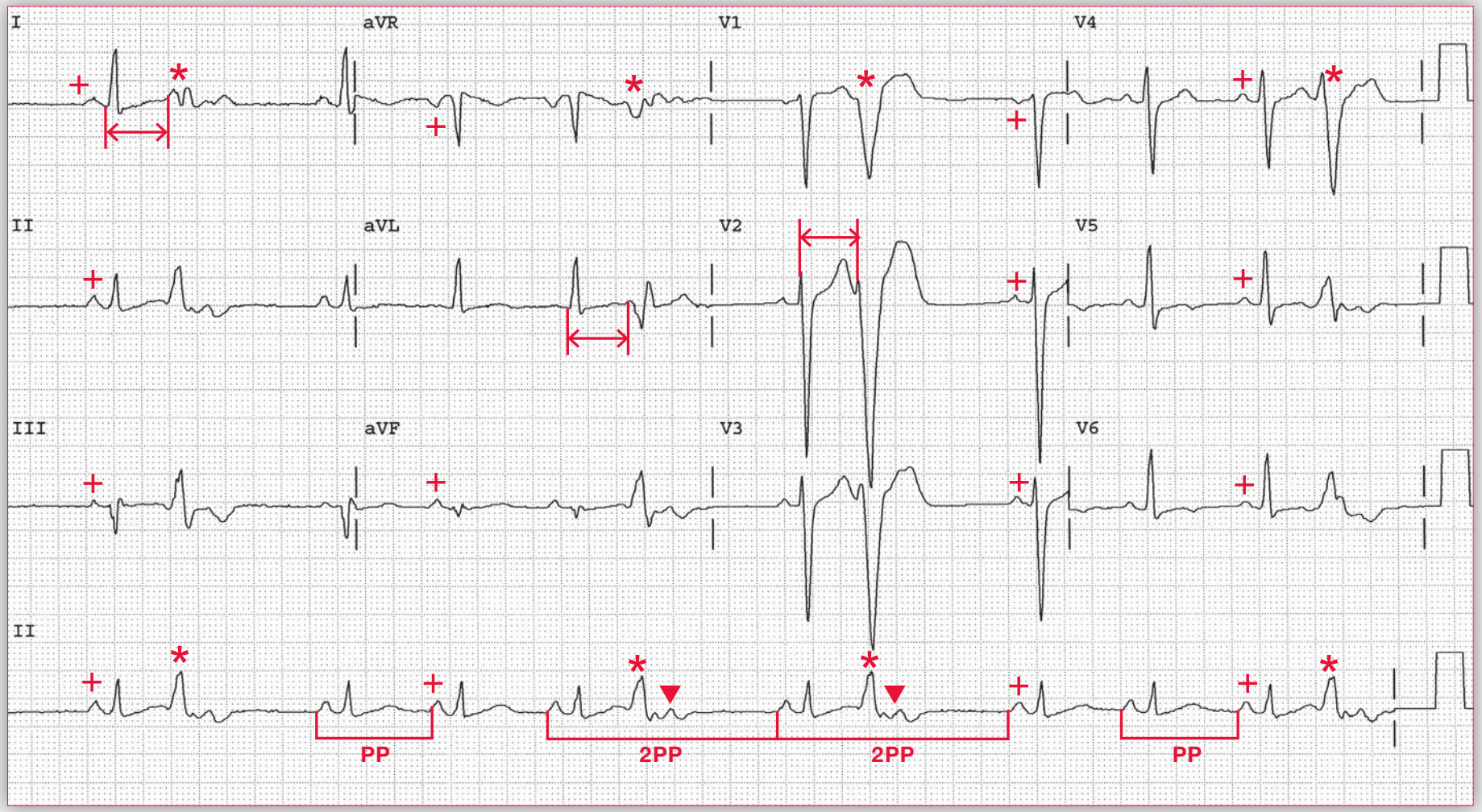
Practice Case 91

A patient on telemetry is noted to have occasional P waves that are not followed by a QRS complex. The patient feels well, but his health care providers are concerned that he may develop high-grade heart block.

Are their concerns founded?

What is occurring?





ECG 91 Analysis: Normal sinus rhythm, unifocal premature ventricular complexes

There is a regularly irregular rhythm as a result of four premature QRS complexes (*) that are followed by a pause. The early QRS complexes (second, sixth, eighth, and 12th complexes) are wide (0.20 sec) and have an abnormal morphology. Although they appear to have a left bundle branch block morphology, with a QS complex in lead V1 and a broad R wave in leads I and V6, the morphology has a atypical features that are not seen with a left bundle branch block (*ie*, prominent notching and broad Q wave in lead aVL). There is no P wave before any of these premature complexes; they are premature ventricular complexes. They all have the same morphology and hence are unifocal. There is a fixed coupling interval (↔) between the premature complex and the preceding QRS complex. The narrow QRS complexes are regular with a normal duration (0.10 sec) and morphology. There is a physiologic left axis, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). There is a P wave (+) before each narrow QRS complex with a fixed PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm at a rate of 76 bpm. The QT/QTc intervals are normal (400/450 msec).

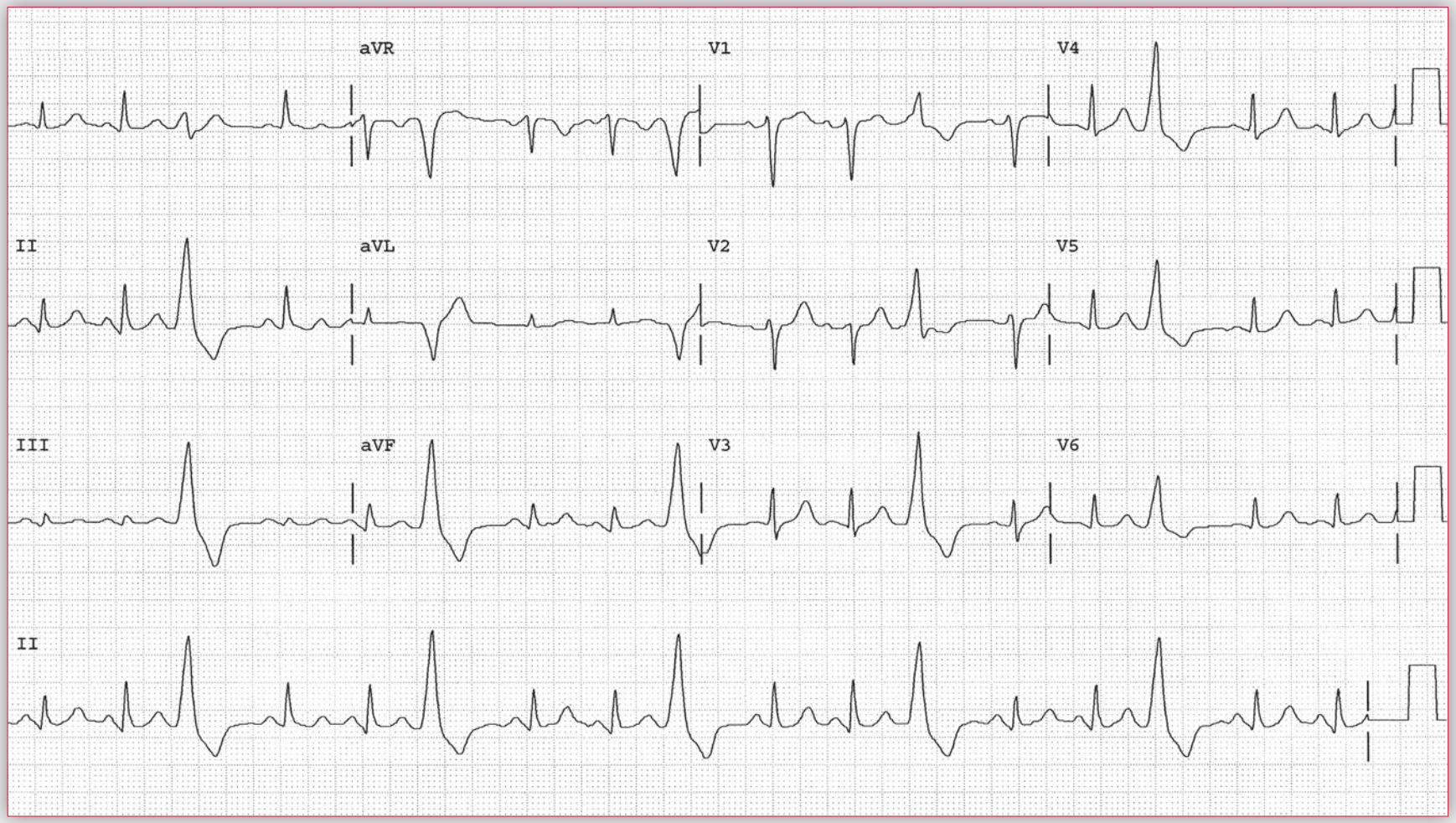
The PP interval around the premature ventricular complex is twice the underlying sinus rate (PP interval) (⊔); that is, there is a compensatory pause. However, an on-time sinus P wave (▼) can be seen after each premature ventricular beat. This P wave, however, is nonconducted (*ie*, it is not followed by a QRS complex). This is due to the fact that the AV node is completely refractory as a result of retrograde conduction through the node resulting from the premature complex. Hence the on-time sinus activity is not conducted antegradely through the AV node, resulting in an on-time but nonconducted sinus P wave and a full compensatory pause. These nonconducted P waves do not indicate intrinsic conduction disease but rather a normal physiologic effect of premature ventricular impulses blocking the conduction of the sinus P-wave impulse. ■

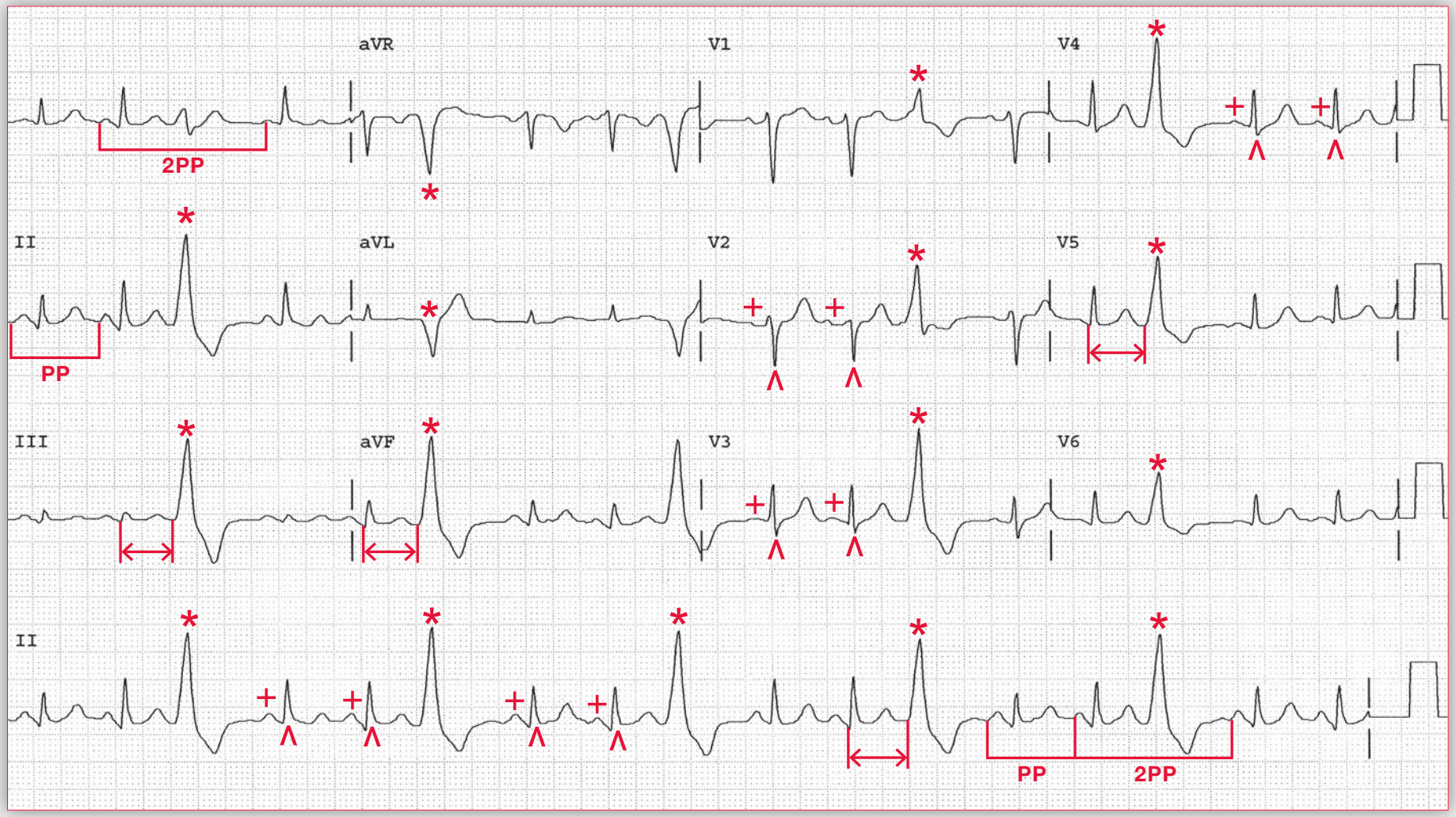
Notes

Practice Case 92

A patient is noted to have a regularly irregular pulse on exam. An ECG is obtained.

What is the explanation for this unusual pulse pattern?





ECG 92 Analysis: Sinus rhythm, premature ventricular complexes in a trigeminal pattern

The rhythm is regularly irregular as a result of premature complexes. There are two narrow QRS complexes (^) (duration 0.08 sec), each of which is preceded by a P wave (+) with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence, these are sinus complexes. The axis of the sinus QRS complexes is normal, between 0° and +90° (positive QRS complex in leads I and aVF). These QRS complexes have a normal morphology. The QT/QTc intervals are normal (350/450 msec). The PP interval is stable, and the rate is 100 bpm. After the two sinus complexes is a premature complex (*) that is wide (duration 0.18 sec) and has an abnormal morphology. Although there is a tall R wave in lead V1, the morphology is not typical for a right bundle branch block as there are no terminal S waves in leads V5-V6. More importantly, there is positive concordance (*ie*, tall R waves in leads V1-V6). Positive concordance does not occur as a result of conduction through the normal His-Purkinje system but rather represents direct myocardial activation as occurs with a ventricular complex due to Wolff-Parkinson-White or a paced complex. The QRS complex is not preceded by a P wave. These are premature ventricular complexes (PVCs). The coupling interval (↔) between the second narrow complex and the PVC is fixed, indicating that the PVC is the result of reentry. Because the PVCs are single, the impulse travels around the circuit only once. Every third QRS complex is a PVC, so this is termed ventricular trigeminy. Each of the PVCs has the same morphology (*ie*, they are unifocal). The PVC is followed by a compensatory pause; that is, the PP interval around the PVC is equal to two sinus (PP) intervals (⊔). This is due to the fact that premature impulse enters the AV node retrogradely, causing it to be refractory and unable to conduct the next on-time sinus P wave. The next sinus P wave is conducted normally. ■

Notes

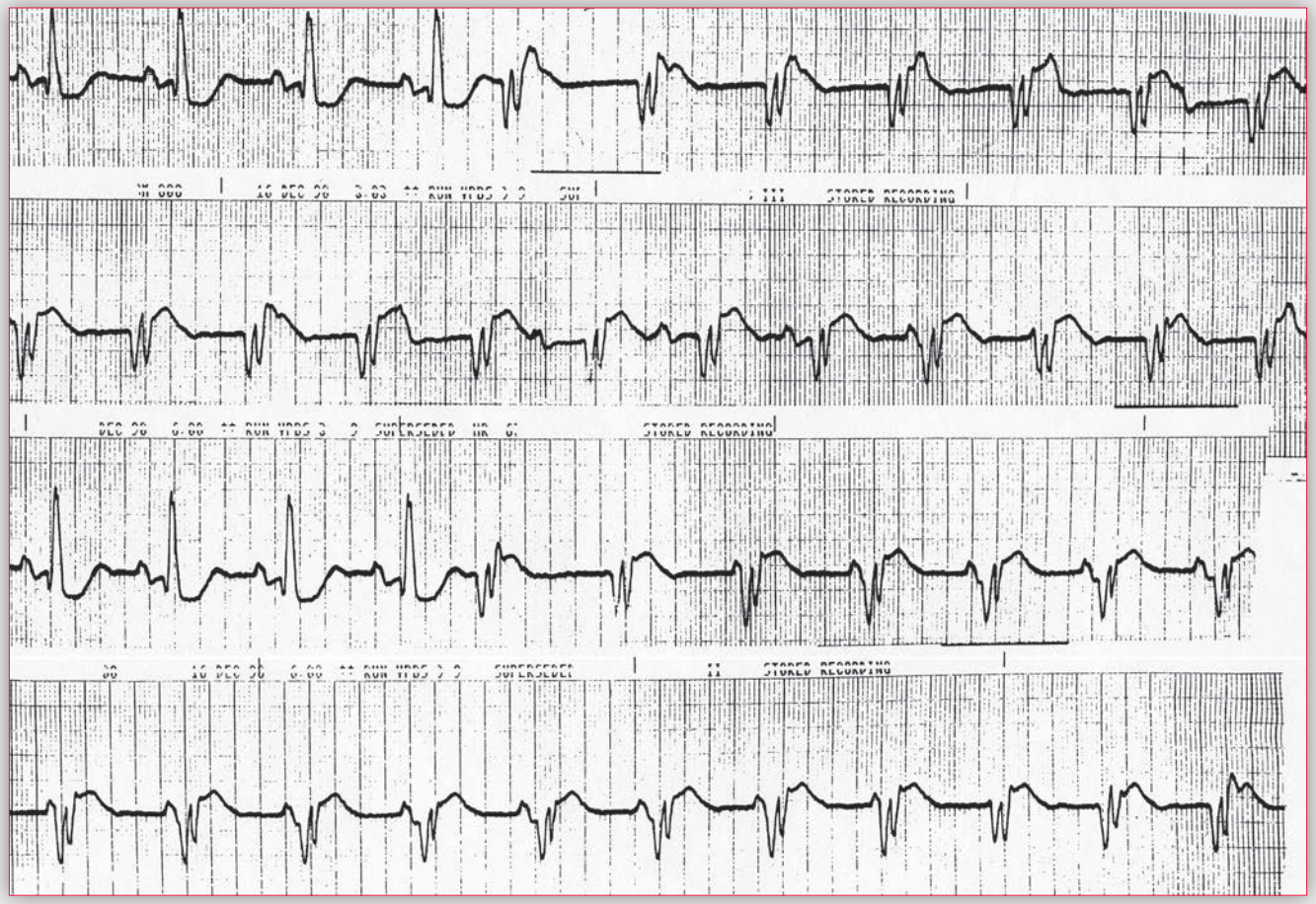
Practice Case 93

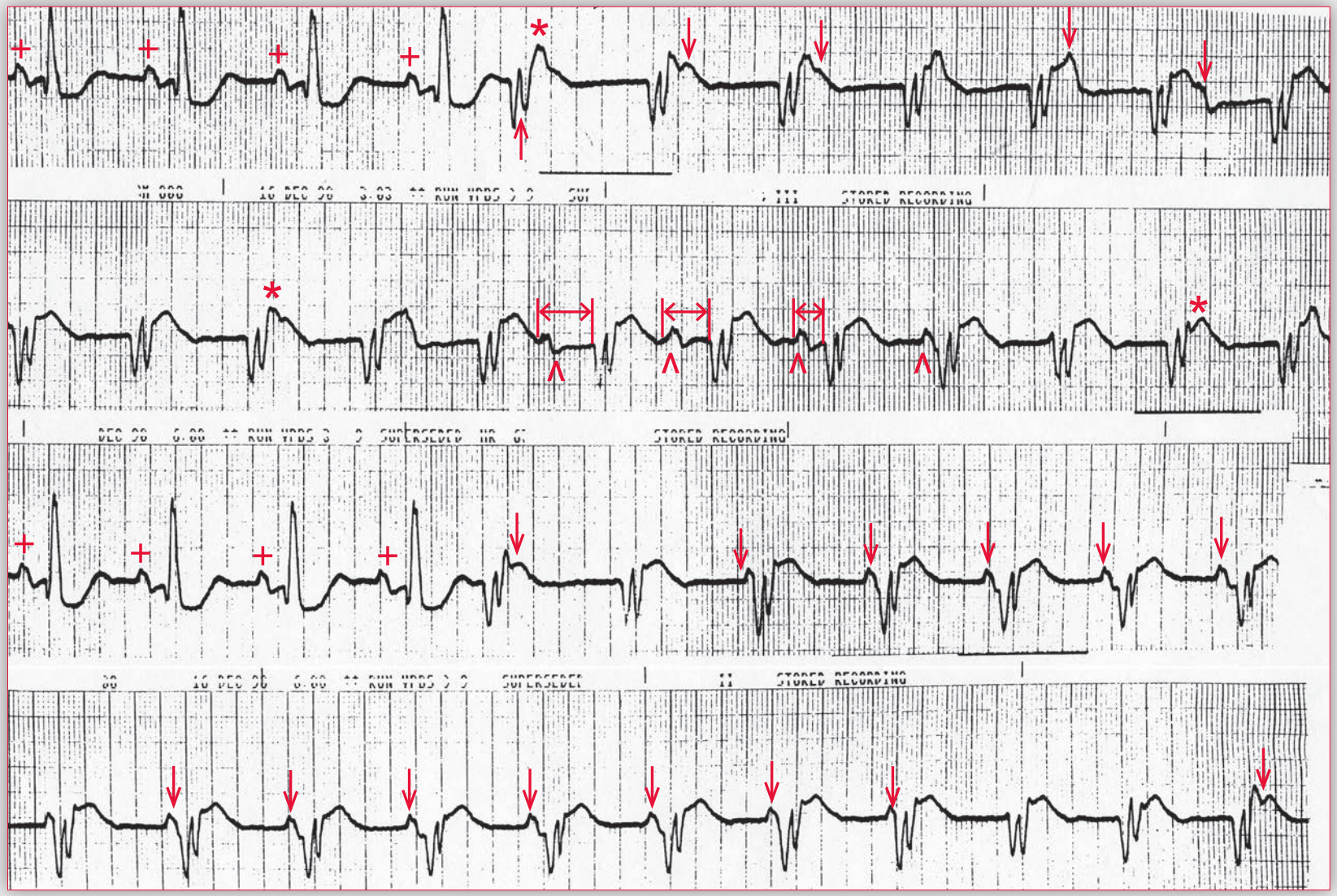
A 77-year-old woman with a history of heart failure and chronic kidney disease presents with intermittent lightheadedness and throbbing sensations in her neck. She has dilated cardiomyopathy with a left ventricular ejection fraction of 35% and was recently started on digitalis for significant heart failure symptoms (New York Heart Association class III). You obtain the following rhythm strips while the patient is symptomatic.

What is the rhythm disturbance?

What in particular is causing the throbbing in her neck?

What do you expect to find on physical examination?





ECG 93 Analysis: Sinus rhythm, AV dissociation with accelerated idioventricular rhythm (AIVR)

This is a series of rhythm strips. The first two are continuous, as are the second two. Noted on the first strip are four regular QRS complexes with a slightly prolonged duration (0.12 sec), each of which is preceded by a P wave (+) with a constant PR interval (0.20 sec). This is, therefore, a sinus rhythm at a rate of 60 bpm. However, the fifth QRS complex (↑), which occurs early, has a different morphology and is not preceded by a P wave. By measuring the PP intervals, however, it can be seen that there is an on-time P wave at the end of the fifth QRS complex (*), giving the appearance of an R′ waveform. The subsequent QRS complexes, which have the same morphology as the fifth complex, are regular at a rate of 70 bpm. Although P waves are not obvious, there are irregularities of the T waves and ST segments due to embedded or superimposed P waves (↓). Noted on the second strip are regular P waves (^), at a rate of 60 bpm, identical to the initial sinus rate. The P waves are dissociated from the QRS complexes as there is variability of the PR interval (↔), and occasionally the P wave is superimposed on or within the QRS complex. Hence there is AV dissociation. As the atrial rate is slower than the ventricular rate, this is an accelerated idioventricular ventricular rhythm (AIVR).

The bottom two strips show the same pattern of four QRS complexes preceded by a P wave (+) and a constant PR interval followed by regular QRS complexes that are different from the sinus QRS complex and P waves (↓) that are dissociated. The atrial rate is equivalent to the ventricular rate (*ie*, 62 bpm). Hence this is an idioventricular rhythm with isorhythmic dissociation; that is, AV dissociation is present, but the atrial and ventricular rates are identical.

Given the patient's history of chronic renal disease, this rhythm is very possibly the result of toxic effects of the recently initiated digitalis. Digitalis toxicity is associated with depression of the normal pacemaker tissue (sinus and AV nodal) by enhanced vagal tone as well as augmentation of outputs from the central sympathetic nervous system. Hence as sinus and AV nodal depression occur, the acceleration of a myocardial focus by sympathetic stimulation results in an AIVR.

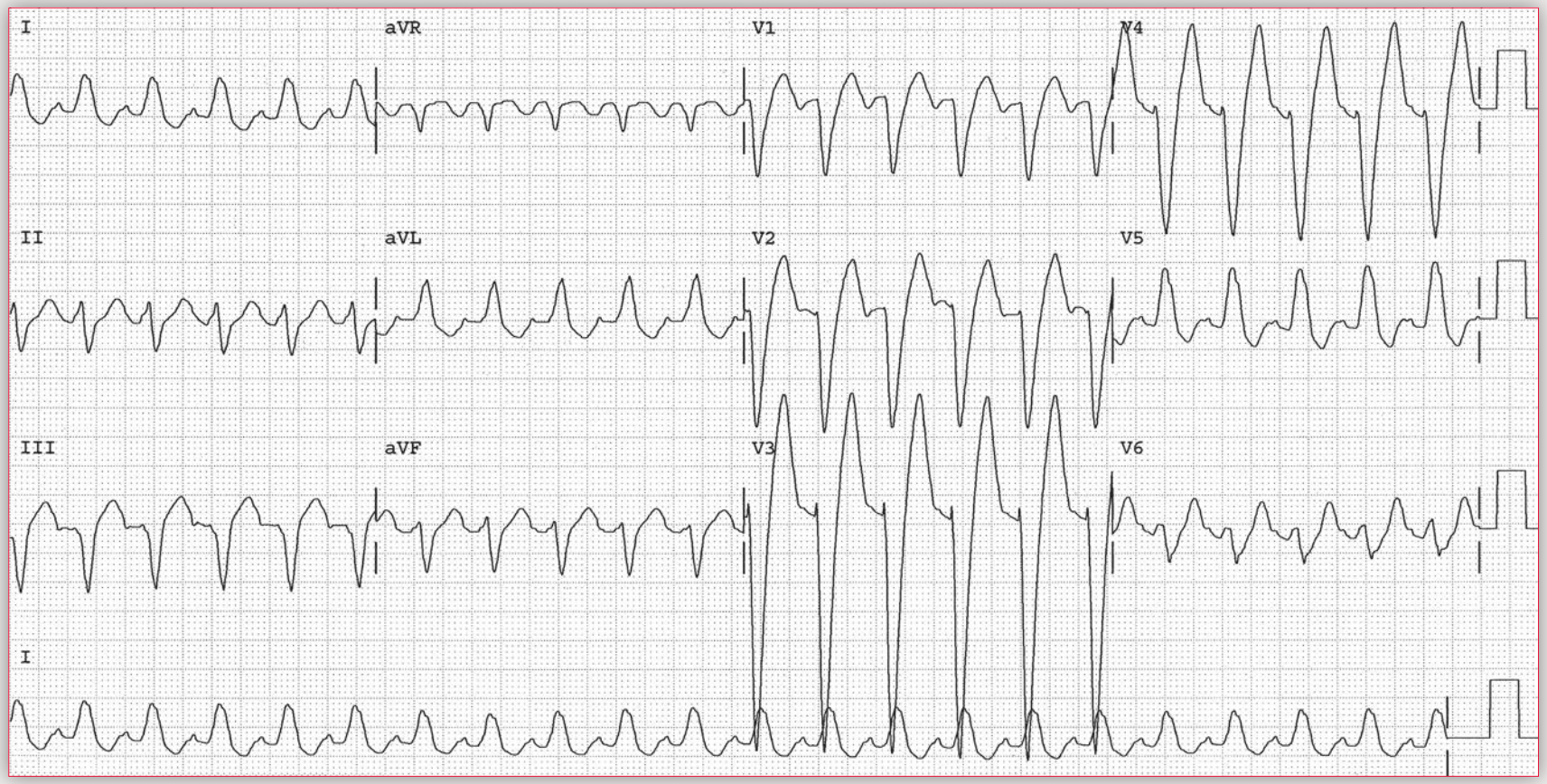
Common physical exam findings associated with AV dissociation and AIVR include the following:

- Variable intensity of the peripheral pulse due to changes in the relationship between atrial and ventricular contraction and hence beat-to-beat variability in stroke volume
- Cannon A waves, resulting from occasional atrial contraction against a closed tricuspid valve (The cannon A waves can be associated with throbbing sensations in the neck.)
- Variable intensity of S1 due to varying degrees of mitral and tricuspid valve closure at the time of ventricular systole ■

Practice Case 94

A 63-year-old man with diabetes, hypertension, and 6 months of intermittent exertional substernal chest pressure undergoes an exercise stress test. An ECG is obtained at peak exercise (ECG 94A). A second ECG (94B) is obtained the following day.

ECG 94A

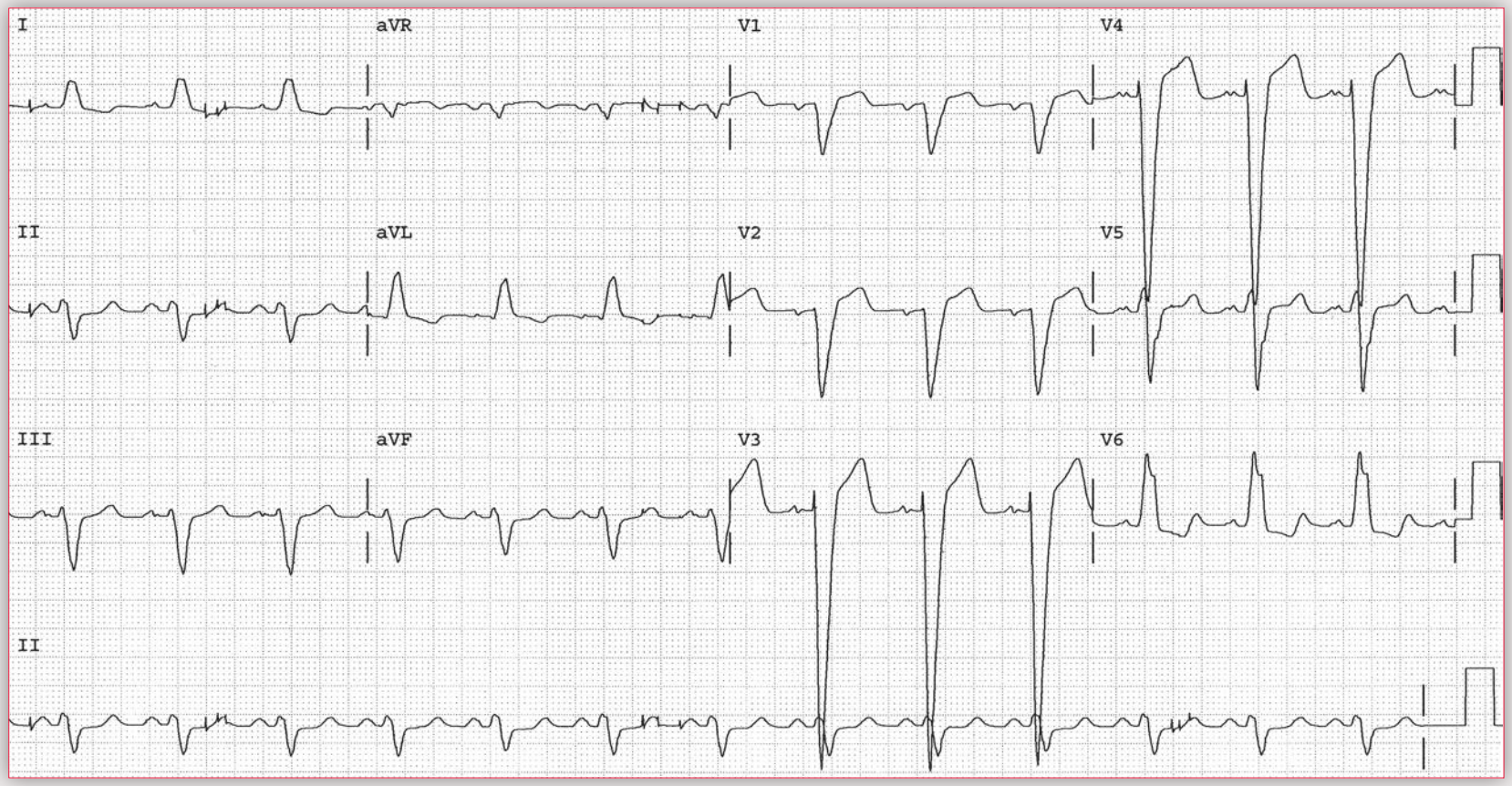


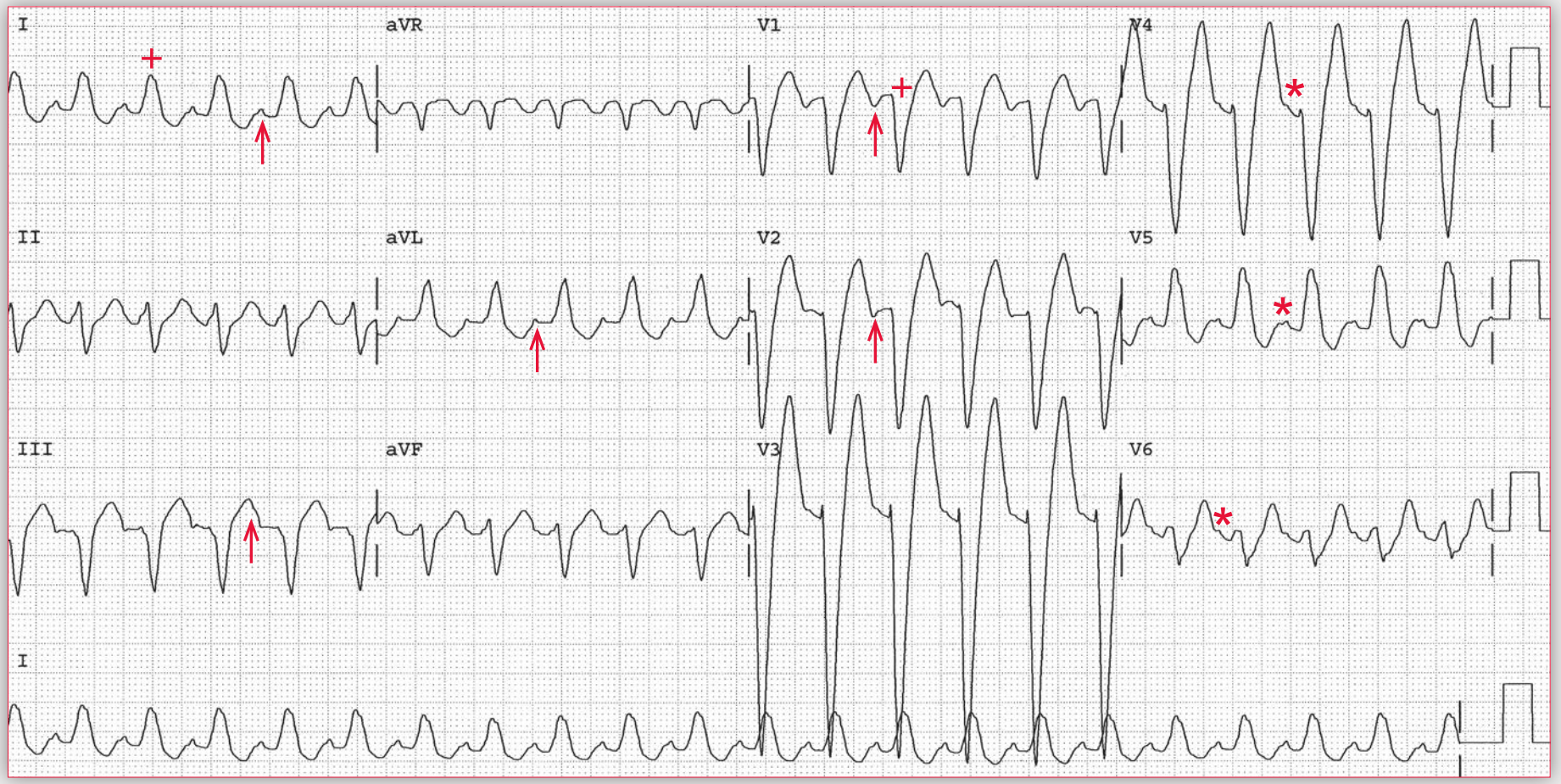
Practice Case 94

What is the underlying rhythm?

Does the patient have coronary artery disease?

ECG 94B





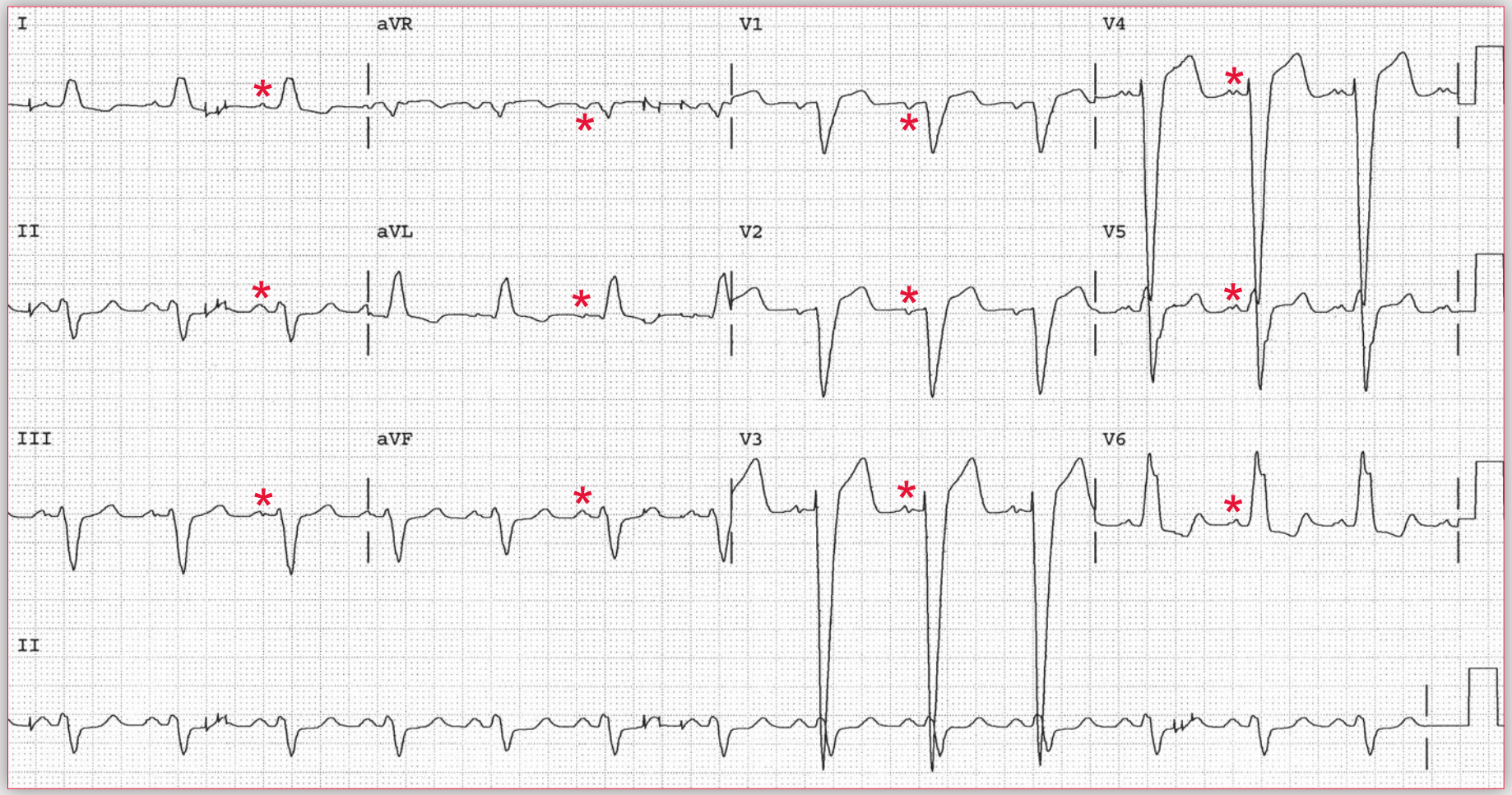
ECG 94A Analysis: Sinus tachycardia, left bundle branch block

ECG 94A shows a regular rhythm at a rate of 130 bpm. P waves are not obvious in most leads. However, a positive P wave can be seen before each QRS complex in leads V4-V6 (*). The PR interval is stable (0.14 sec). Using this PR interval, it can be seen that the negative deflection noted in leads V1-V2 (↑) as well as the positive deflection (↑) in leads I, aVL, and III are the P waves. This is, therefore, sinus tachycardia.

The QRS complex duration is prolonged (0.16 sec), and there is a pattern characteristic of left bundle branch block (LBBB; broad R wave in leads I and V5 and a QS complex in lead V1 [+]). The axis in the frontal plane is leftward (positive QRS complex in lead I and negative QRS complex in leads II and aVF). The QT/QTc intervals are prolonged (320/470 msec) but are normal when corrected for the prolonged QRS complex duration (240/360 msec).

In the presence of LBBB, an ECG stress test cannot be interpreted because ST-segment changes cannot be analyzed. With LBBB, left ventricular activation is no longer via the His-Purkinje system but rather is the result of direct myocardial activation. Therefore, abnormalities of the left ventricle, including ischemia, cannot be evaluated. With LBBB, the presence of any ST-segment changes with exercise are not reliable for the diagnosis of myocardial ischemia or flow-limiting coronary artery disease. Ischemic evaluation in patients with LBBB must include either myocardial perfusion imaging (*ie*, single-photon emission computed tomography) or stress echocardiography to assess for inducible wall motion abnormalities. Other situations in which the ECG stress test cannot be used to interpret ST-segment changes include ventricular pacing and pre-excitation syndrome. These are conditions in which activation of the left ventricle does not occur via the normal His-Purkinje system but is the result of direct ventricular activation.

continues



ECG 94B Analysis: Normal sinus rhythm

ECG 94B, obtained the following day, shows a regular rhythm at a rate of 80 bpm. As a result of the slower sinus rate, the P waves (*) are now obvious before each QRS complex and the PR interval is stable (0.16 sec). Comparing the PR interval on this ECG, the P wave can be further identified on **ECG 94A** and the negative waveform seen in leads V1-V2 is confirmed to be the P wave.

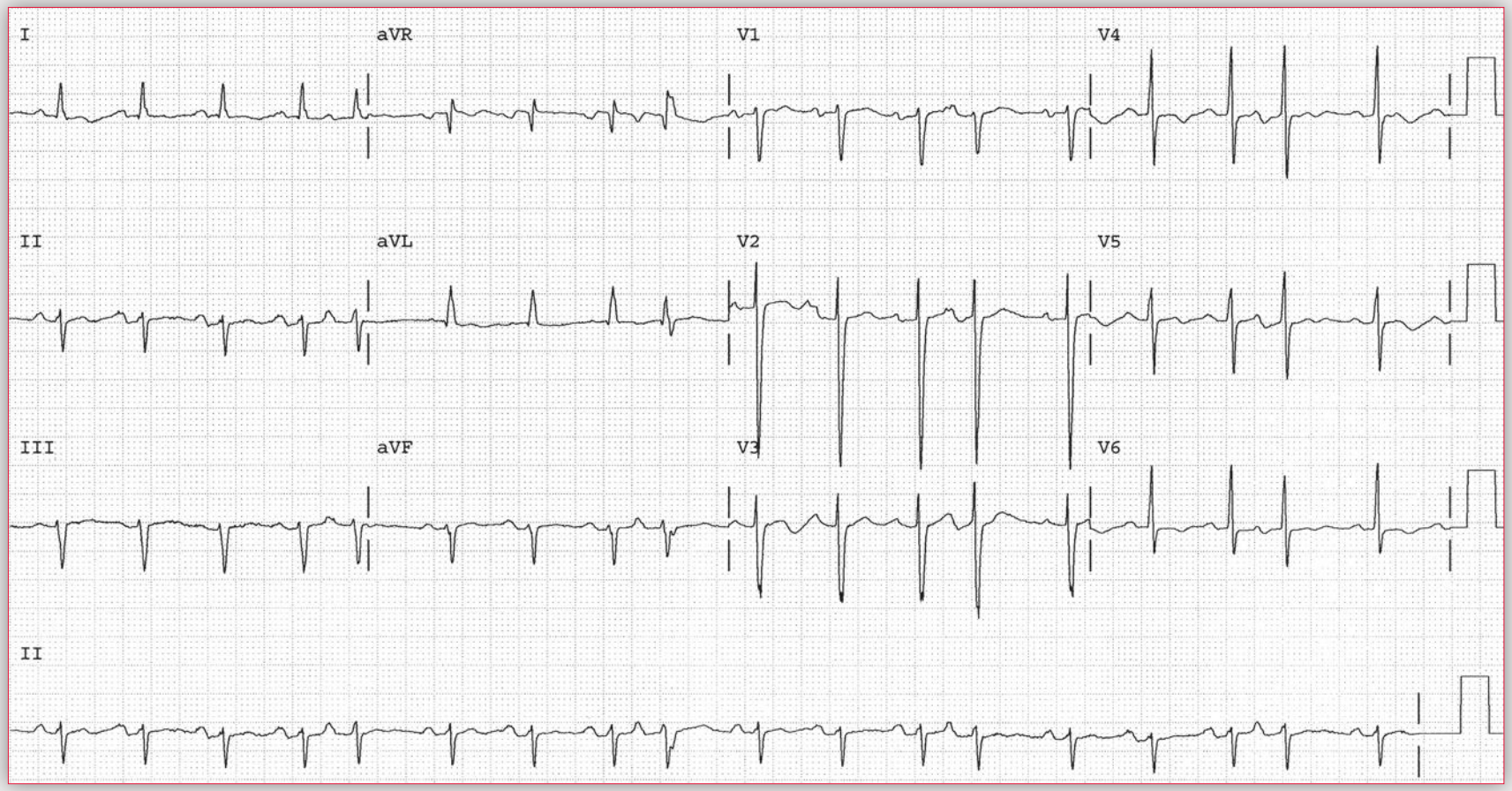
The QRS complex duration is prolonged (0.16 sec) and has the morphology of an LBBB (broad R wave in leads I and V6 and a QS complex in lead V1). The QRS complex morphology and QT/QTc intervals are the same as those in **ECG 94A**. This confirms the fact that the rhythm in **ECG 94A** is sinus tachycardia with an underlying LBBB. ■

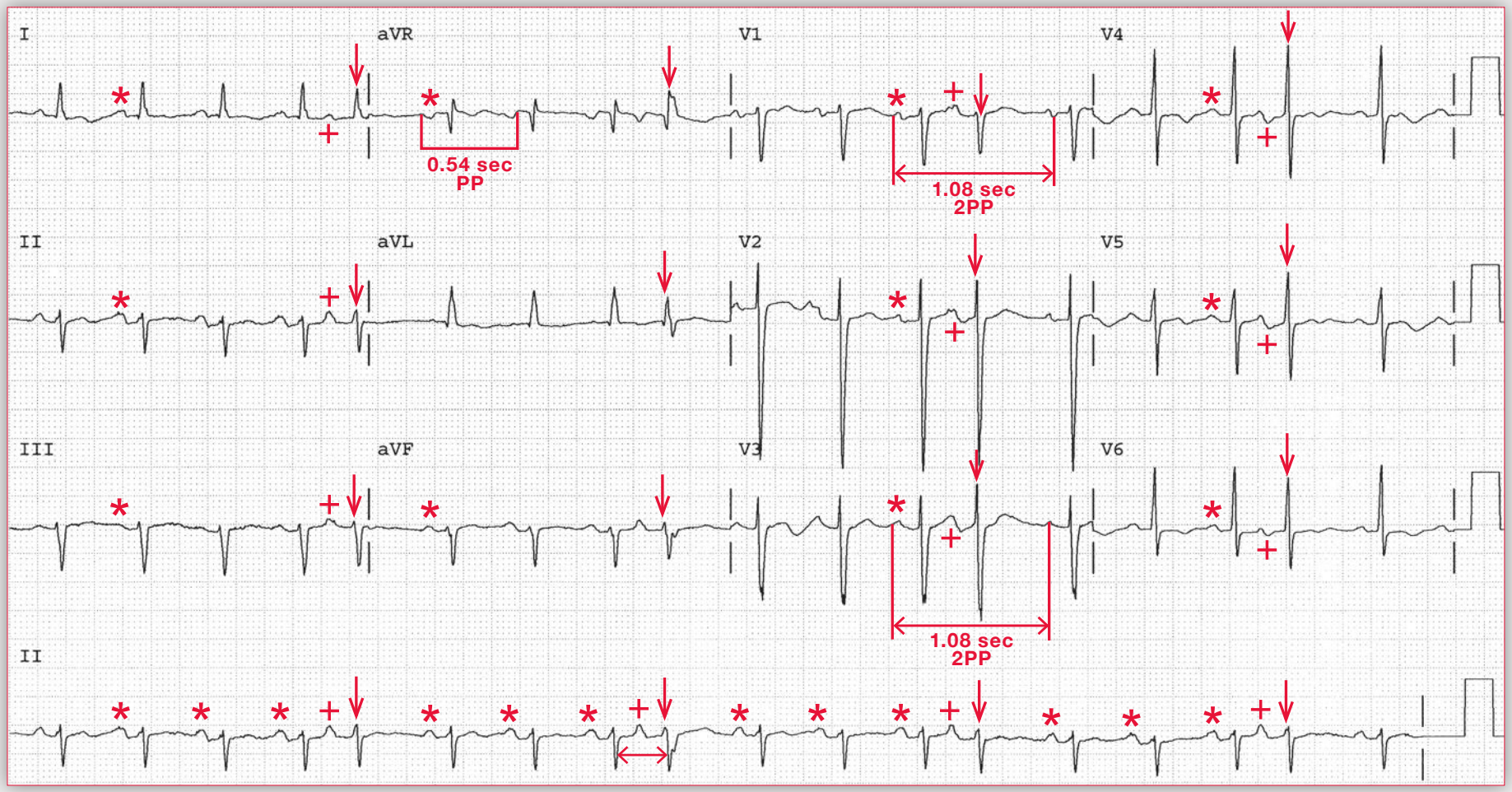
Notes

Practice Case 95

A 32-year-old man with depression and alcohol dependence is brought to the emergency department by his friends, who found him unconscious at home. His friends noted multiple empty beer bottles laying next to him. The man's serum alcohol level is elevated, but the remainder of a toxicology screen is normal. The following ECG is obtained.

What is the rhythm abnormality?





ECG 95 Analysis: Sinus tachycardia with premature atrial beats (unifocal), left anterior fascicular block

The rhythm is irregular, but there is a regular pattern (*ie*, the short, intermediate, and long RR intervals are the same). Hence this is a regularly irregular rhythm. When the rhythm is regular (rate of 102 bpm) there is a P wave (*) before each QRS complex and the PR interval is constant (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia.

The early beats (↓) are also preceded by a P wave (+) with a morphology that is different from the sinus P wave. In addition, there is a slightly longer PR interval (0.20 sec). These are premature atrial complexes (PACs), and they are unifocal (all the premature P waves have the same morphology). There is a pause after the PAC and the PP interval surrounding the pause (↔) is less than two sinus PP intervals (⊔). Hence it is less than a full compensatory pause.

The QRS complex duration (0.08 sec) and morphology are normal. The QT/QTc intervals are normal (280/380 msec). The axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). There are two etiologies for an extreme left axis: an old inferior wall myocardial infarction in which there is a deep initial Q wave in leads II and aVF or a left anterior fascicular block in which the QRS complex in leads II and aVF has an rS morphology. Hence this is a left anterior fascicular block. The left bundle, which innervates the left ventricle, divides into two major branches: a left anterior and left posterior fascicle. There is also a third branch known as a median fascicle or septal branch. When there is a complete block of impulse conduction through the left anterior fascicle, all left ventricular activation is via the left posterior fascicle and the impulse is directed up and to the left, accounting for the extreme left axis. When conduction within the left posterior fascicle is blocked, the left ventricle is activated via the left anterior fascicle and the impulse is directed down and to the right; therefore, the axis is rightward.

PACs are common and benign. However, they may be associated with symptoms such as palpitations. This is generally the result of an increased stroke volume that occurs with the post-extrasystolic complex, which is due to the increased left ventricular filling occurring with the long period of diastole. As a result of the larger end-diastolic volume, there is an increase in the force of left ventricular contraction and stroke volume due to the Frank-Starling effect. When the pulse is felt, there is a pause and then a more prominent pulse. This is often referred to as a "skipped beat." The "skipped beat" is due to the fact that the PAC is associated with a reduced stroke volume, which is caused by reduced filling of the left ventricle as a result of the early or premature complex and contraction.

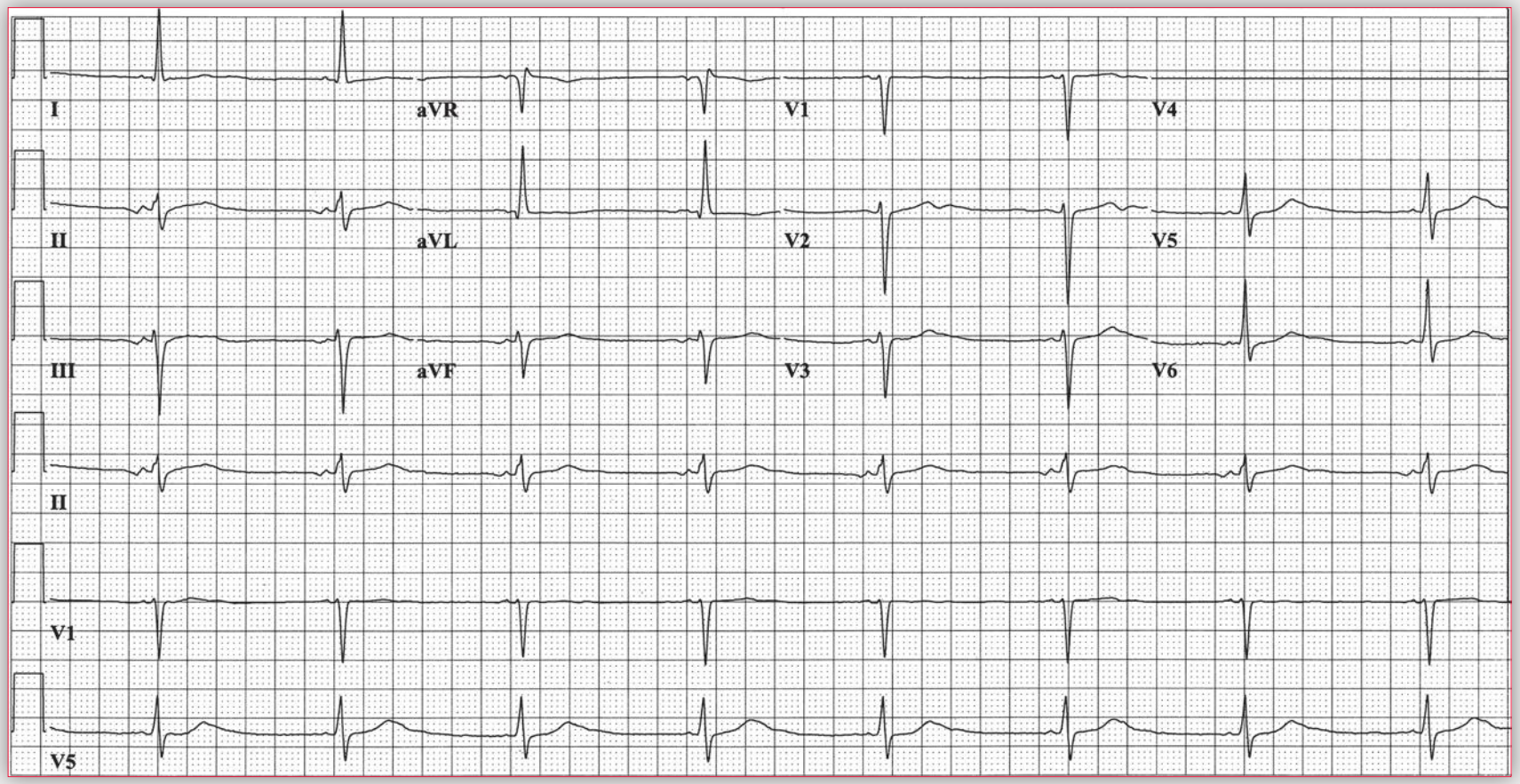
PACs generally do not require therapy. Reassurance is often necessary in patients who are concerned about the palpitations. On occasion, therapy with a β-blocker when symptoms are very pronounced may be effective for reducing left ventricular inotropy, the force of the post-extrasystolic contraction, and hence the sensation of palpitations. ■

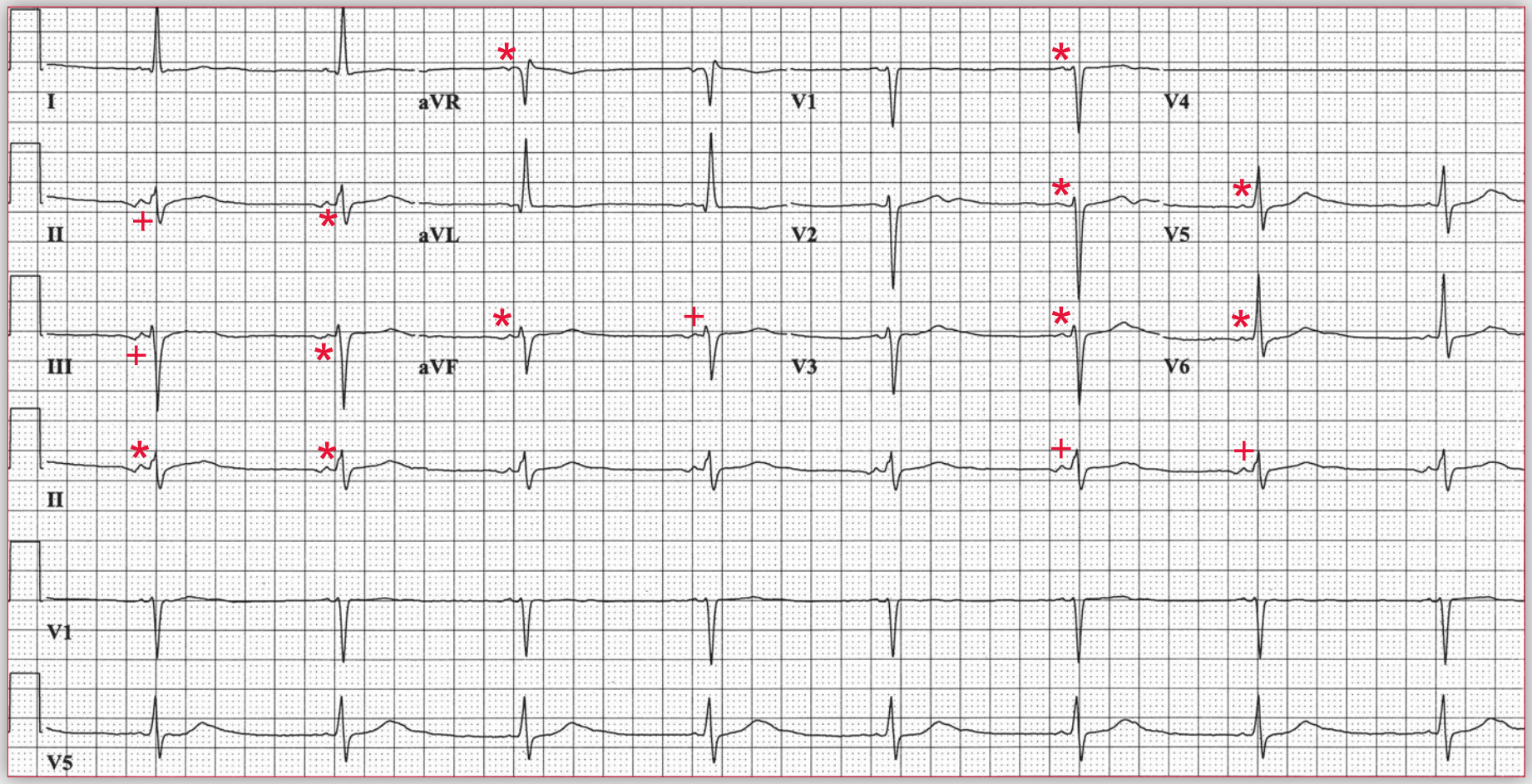
Notes

Practice Case 96

A 56-year-old man is admitted with exertional angina. A myocardial perfusion test shows inducible anterior ischemia. Shortly after the stress test, an ECG is obtained.

What does the ECG show?





ECG 96 Analysis: Ectopic atrial rhythm, left axis

There is a regular rhythm at a rate of 48 bpm. There is a P wave (*) before each QRS complex with a constant PR interval (0.12 sec). However, the P wave is abnormal and is biphasic (negative-positive) in leads II, III, and aVF. The atrial impulse is not originating in the sinus node but is from an ectopic atrial focus. This is, therefore, an ectopic atrial rhythm. The QRS complexes have a normal duration (0.10 sec) and morphology and a physiologic left axis, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (480/430 msec).

An atrial rhythm is seen in patients with no cardiac pathology. However, it may also be seen with many types of cardiac pathology or other situations affecting the heart, including myocardial infarction, pulmonary decompensation, infection, alcohol excess, hypokalemia, hypoxia, and myocardial stimulants such as cocaine or sympathomimetic agents. Atrial tachycardia may be due to an enhanced ectopic focus that generates an impulses faster than the sinus node or may occur as an escape rhythm as a result of a slow sinus rate. ■

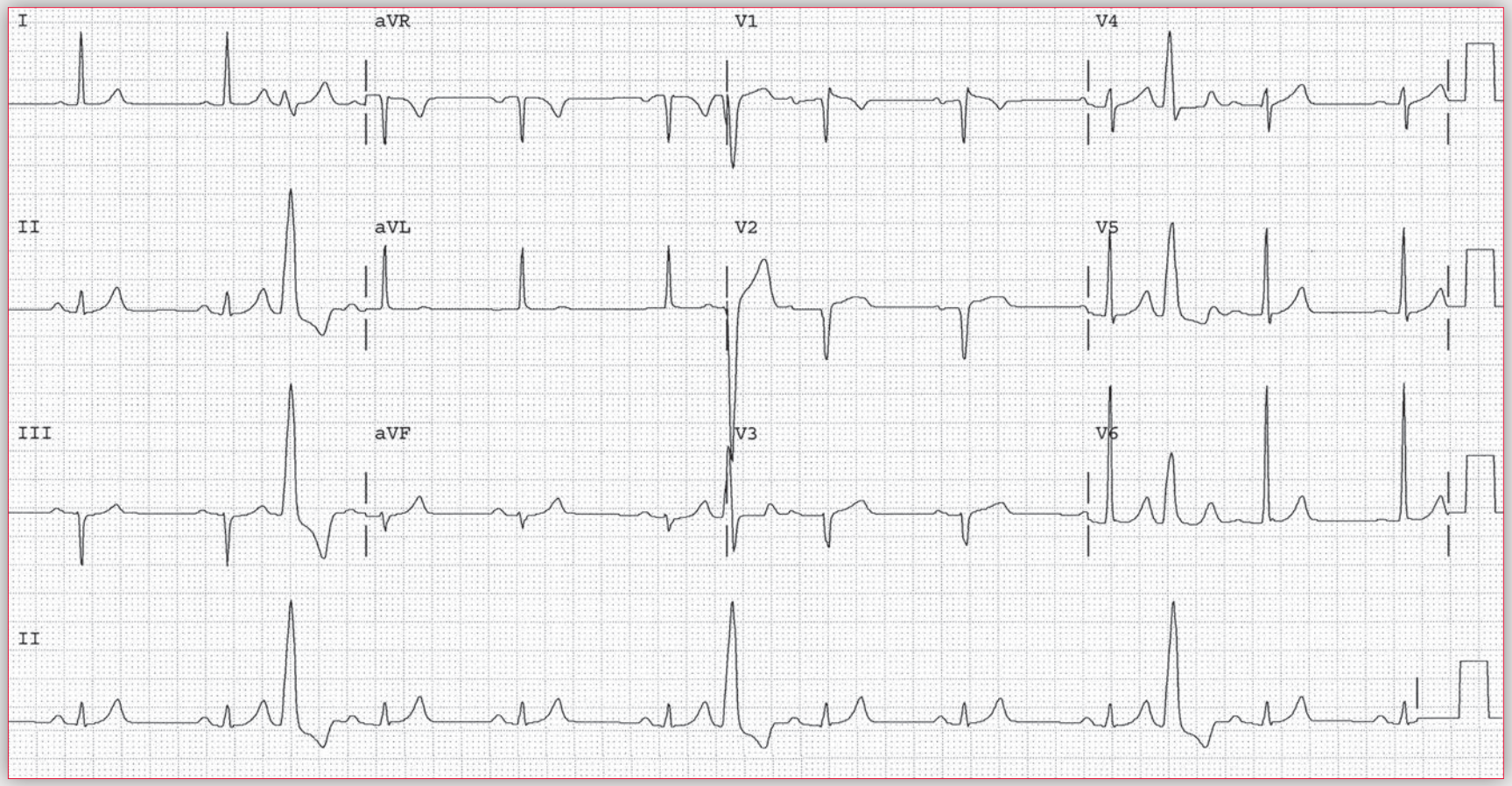
Notes

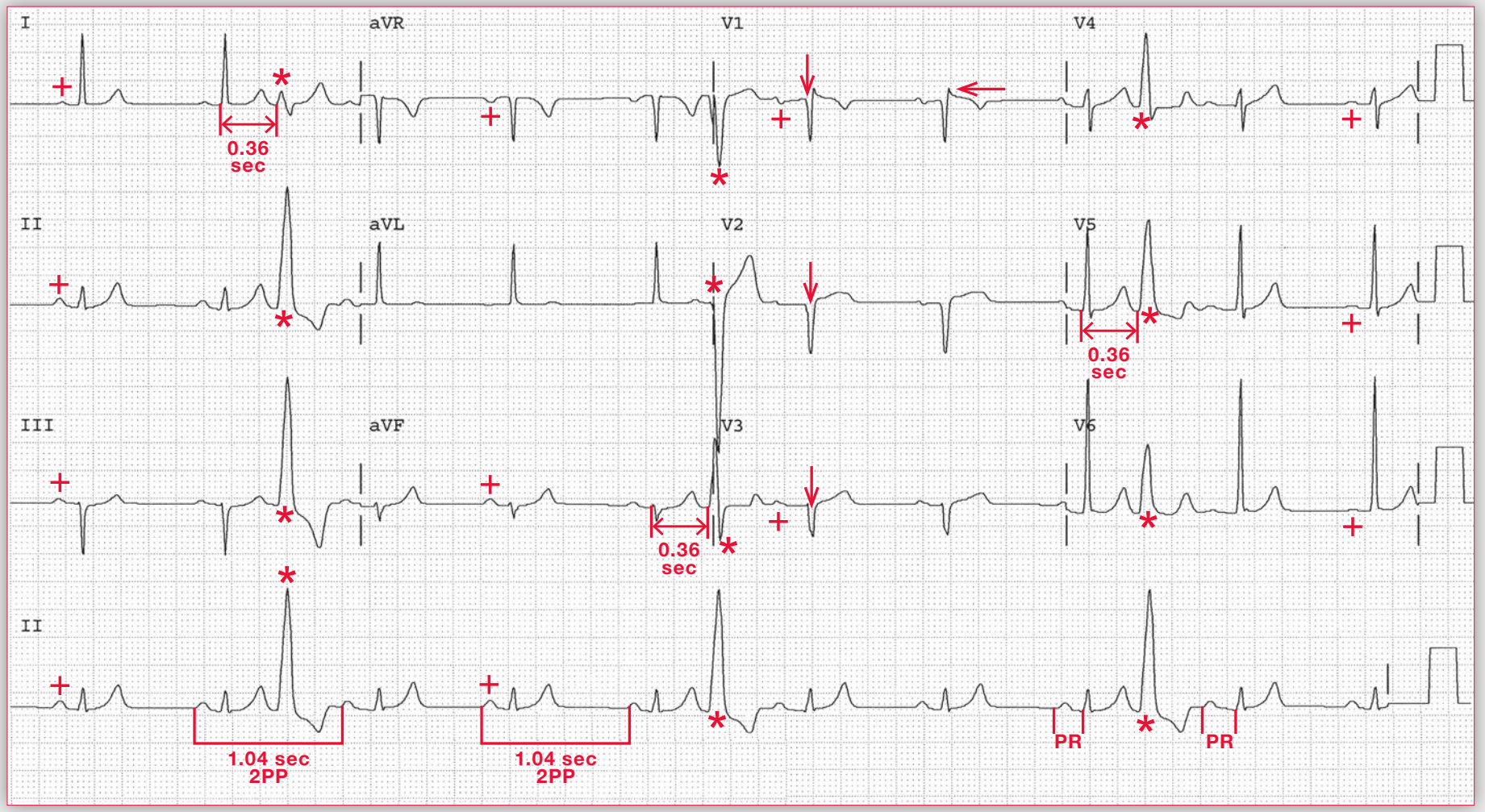
Practice Case 97

A 48-year-old man is noted to have an abnormal ECG during a follow-up visit after admission for a myocardial infarction. The ECG shows a variable PR interval, and the physician suspects that the patient may have some degree of heart block.

Is the physician correct?

What is the best explanation for the patient's variable PR interval?





ECG 97 Analysis: Normal sinus rhythm, interpolated unifocal premature ventricular complexes, old anteroseptal myocardial infarction

The rhythm is regularly irregular as a result of three early and wide QRS complexes (*) that are not preceded by a P wave. These early QRS complexes (the third, seventh, and 11th complexes) have a width of 0.18 second and an unusual morphology. The coupling interval (the interval between the premature complex and the preceding QRS complex) is the same; that is, there is a fixed coupling interval (↔). These are premature ventricular complexes (PVCs). They all have the same morphology and hence are unifocal.

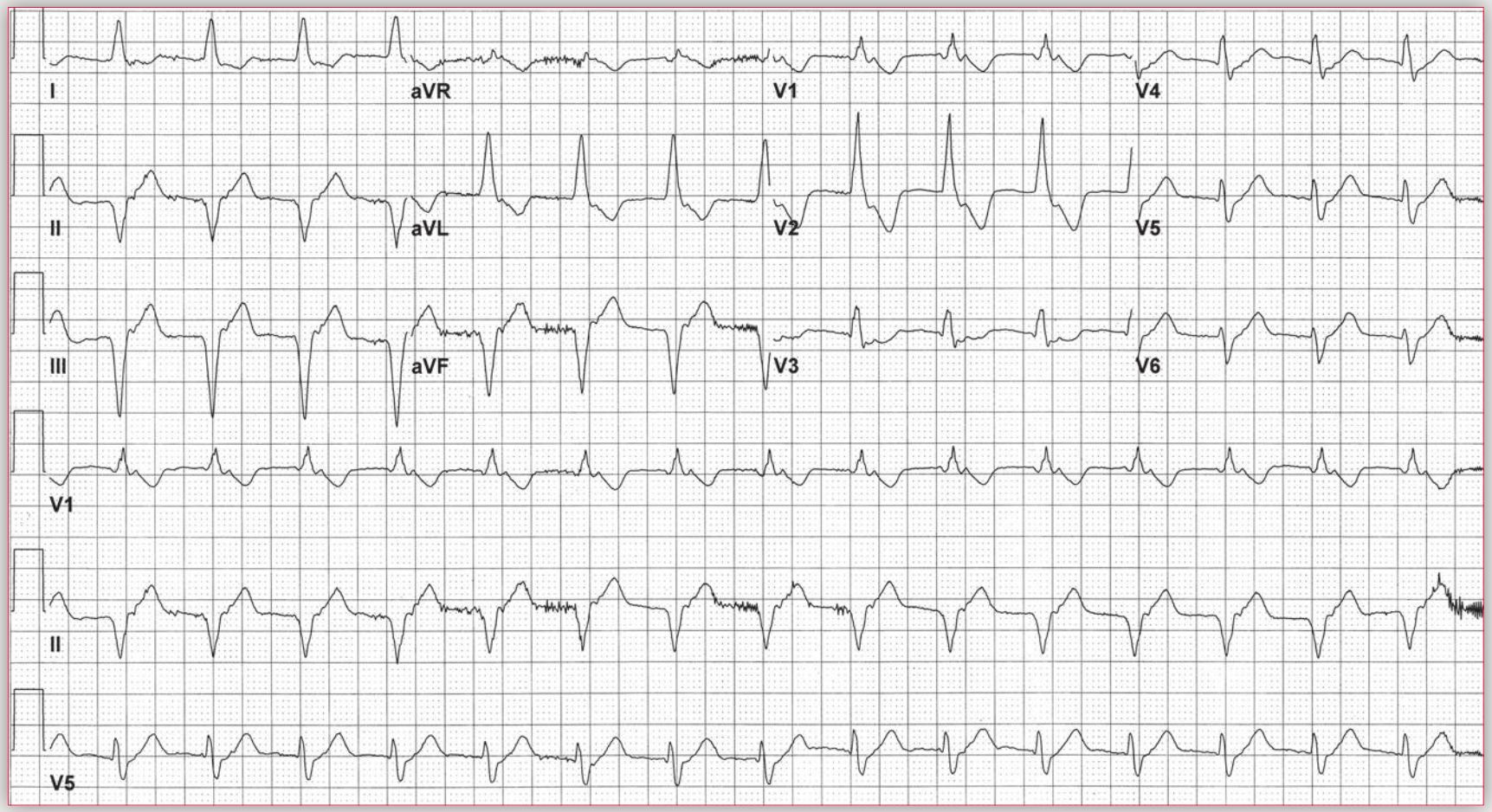
All of the narrower QRS complexes (0.08 sec) are at a regular rate of 62 bpm. The QT/QTc intervals are normal (400/410 msec). They have a physiologic left axis, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). There is a small R′ waveform (←) in lead V1 as a result of a conduction delay to the right ventricle. In addition, there is no septal R wave in leads V1-V3 (↓), which is diagnostic for an old anteroseptal myocardial infarction. There is a P wave (+) before each of the narrow QRS complexes with a stable PR interval (0.20 sec). The P wave is upright in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm.

Noted is that the PP interval surrounding the PVC (⊔) is the same as the underlying sinus rate or the baseline sinus PP interval. Hence these are called interpolated PVCs (*ie*, they do not alter the underlying sinus rate and are not associated with a pause because they do not prevent the on-time sinus P wave from conducting through the AV node). However, the PR interval of the complex following the PVC is longer (0.24 sec) than the baseline PR interval (0.20 sec), even though the P wave is on time. This is the result of retrograde concealed conduction and is frequently seen with interpolated PVCs. The PVC results in retrograde conduction through the AV node. However, an appropriately timed premature beat may result in retrograde conduction that does not completely penetrate the AV node but is extinguished within the node, partially depolarizing it (*ie*, it is concealed within the AV node). In this situation the on-time sinus beat may be conducted through the AV node in an antegrade direction but at a slower conduction velocity as the AV node is partially refractory due to the retrograde conduction from the premature complex. ■

Practice Case 98

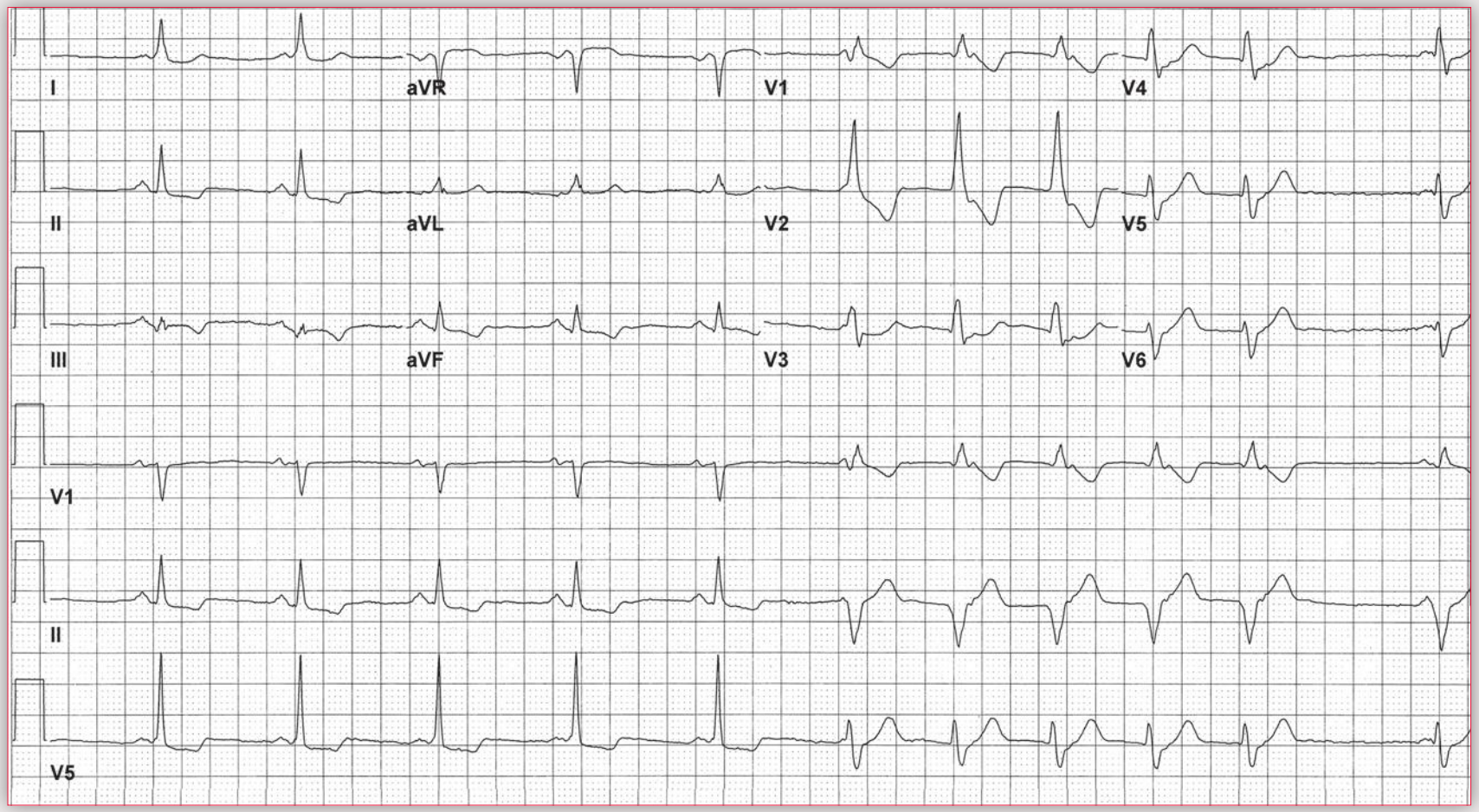
A 48-year-old man presents with acute-onset crushing substernal chest pain, and an ECG shows inferior ST-segment elevations. He is treated urgently with reteplase (a thrombolytic

ECG 98A



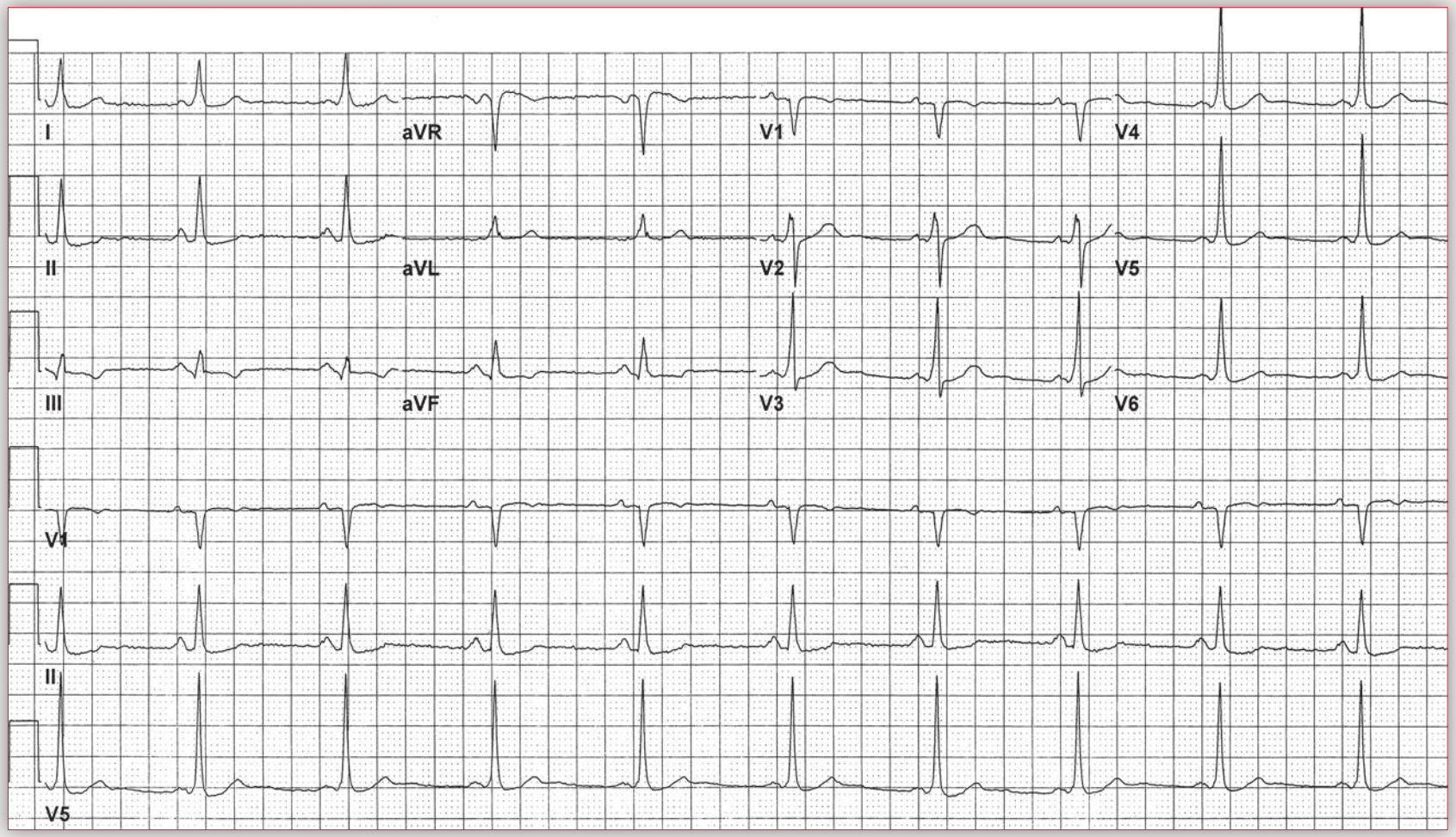
agent), and his symptoms improve. One hour after therapy, two ECGs are obtained (ECG 98A and ECG 98B). On the following day, a subsequent ECG is obtained (ECG 98C).

ECG 98B



Practice Case 98

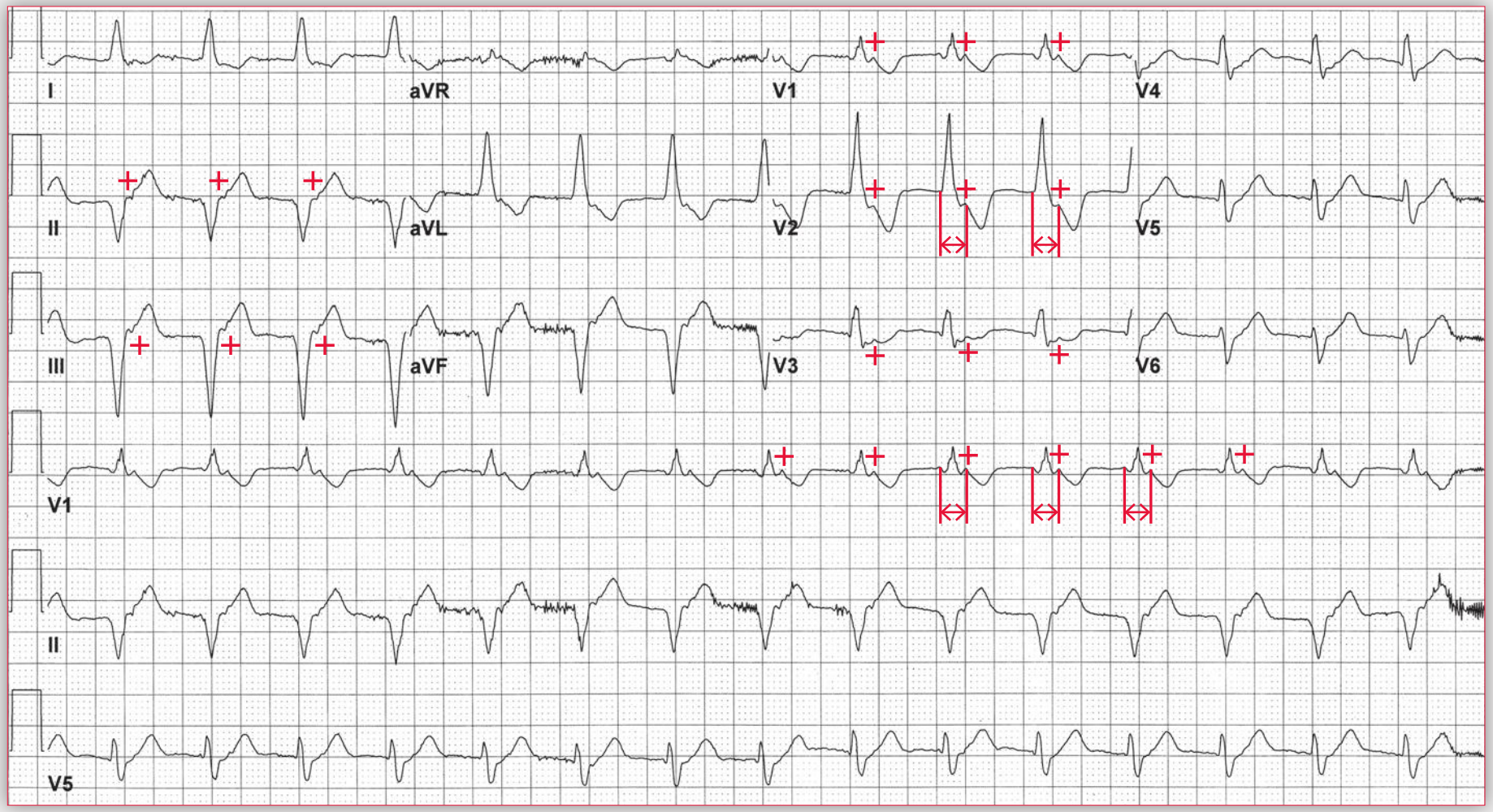
ECG 98C



Practice Case 98

What is the rhythm abnormality?

Does this patient need urgent catheterization?



ECG 98A Analysis: Accelerated idioventricular rhythm (AIVR), retrograde atrial activation

ECG 98A shows a regular rhythm at a rate of 94 bpm. The QRS complexes are wide (0.14 sec) and have an abnormal morphology that is not typical for either a right or left bundle branch block. There are no P waves before any of the QRS complexes, but P waves (+) can be seen after each of the QRS complexes with a fixed RP interval (↔) (0.16 sec). The P waves are best seen as notches on the initial part of the ST segments in leads II, III, aVF, and V1. The QRS complexes are ventricular in origin; therefore, this is an accelerated idioventricular rhythm (AIVR) or slow ventricular tachycardia with retrograde atrial activation.

Ventricular tachycardia is often associated with AV dissociation and an atrial rate that is slower than the ventricular rate. This is due to complete block of antegrade conduction through the AV node as a result of retrograde activation and depolarization of the node by the faster ventricular impulse. Hence the sinus impulse cannot penetrate and conduct through the AV node. When the rate of the ventricular tachycardia is slow, it is possible for the ventricular impulse to conduct completely through the AV node in a retrograde direction and activate the atrium, resulting in a retrograde P wave that follows the QRS complex. In this situation, the retrograde atrial activation suppresses sinus node activity.

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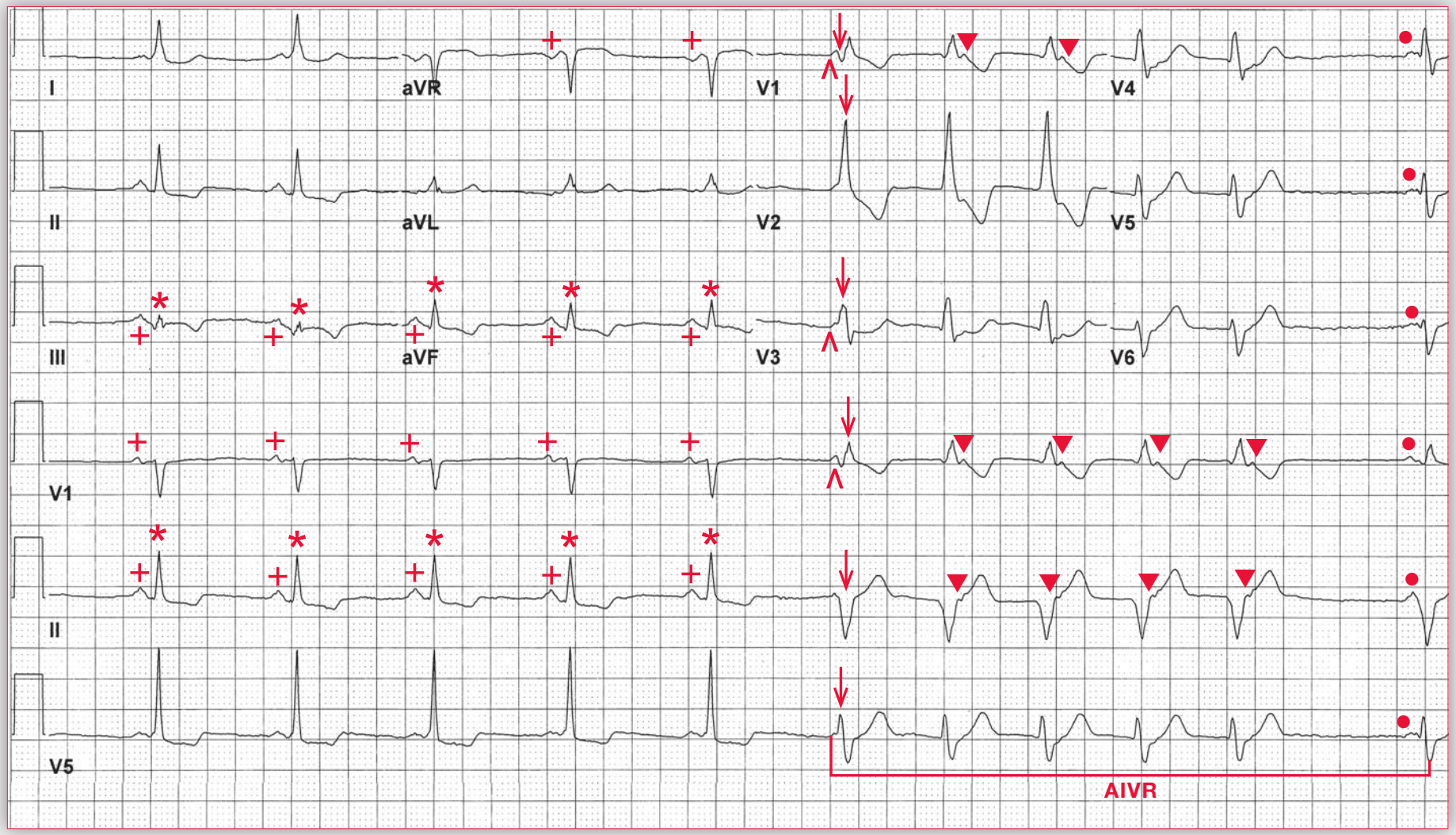

ECG 98B Analysis: Sinus rhythm, AIVR

In **ECG 98B,** the first five QRS complexes (*) have a normal duration (0.08 sec), and there is a P wave (+) before each complex with a constant PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V5; hence these are sinus complexes at a rate of 62 bpm. The QT/QTc intervals are normal (400/410 msec). These five complexes have a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF).

The sixth QRS complex (↓) is early and wide (0.16 sec) and has a different morphology than the first five. This QRS complex resembles those seen in **ECG 98A**. Although there is an on-time sinus P wave (^) preceding this complex, the PR interval (0.08 sec) is shorter than the baseline PR interval and hence the P wave is not conducted. The QRS complexes that follow have the same morphology and duration as the sixth complex, and a P wave (▼) can be seen after the QRS complex (*ie*, notching of the ST segment best seen in the rhythm strips for leads II and V1). The notching in lead II appears to be negative, representing a retrograde and not an antegrade P wave. The QRS complexes are identical to those seen in **ECG 98A** and hence they are ventricular at a rate of 90 bpm. This is an AIVR or slow ventricular tachycardia. The last QRS complex is also ventricular. Although there is a P wave (●) before this complex, the PR interval is shorter (0.10 sec) than the baseline PR interval and hence this is a nonconducted P wave.

AIVR often results from coronary reperfusion after a myocardial infarction. It has been reported that the occurrence of an AIVR after thrombolytic therapy is generally considered to be a marker of successful reperfusion. The rhythm is typically transient and does not require therapy. Thus, cardiac catheterization is not urgently indicated in this patient.

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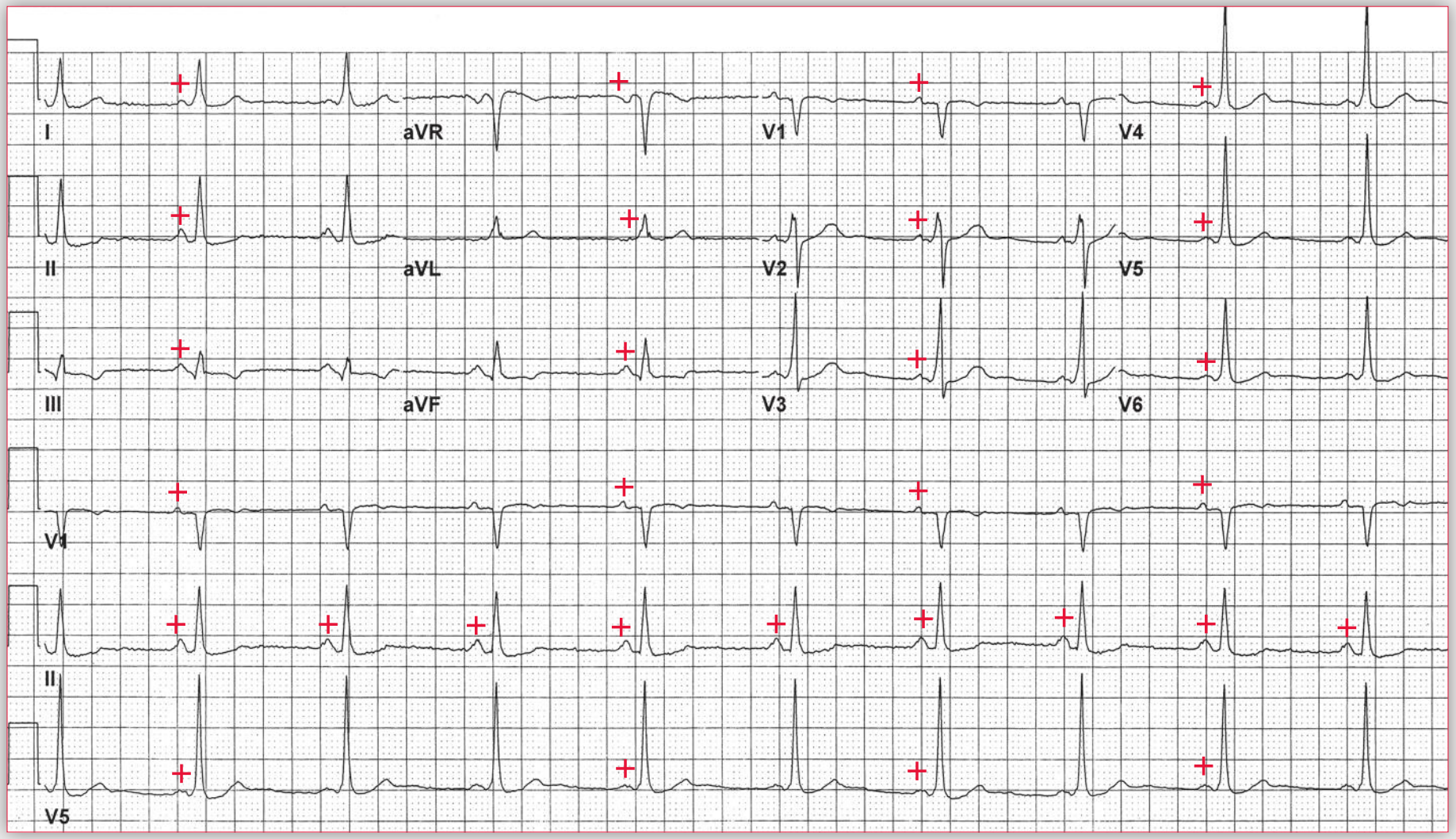


ECG 98C Analysis: Normal sinus rhythm, nonspecific ST-segment changes

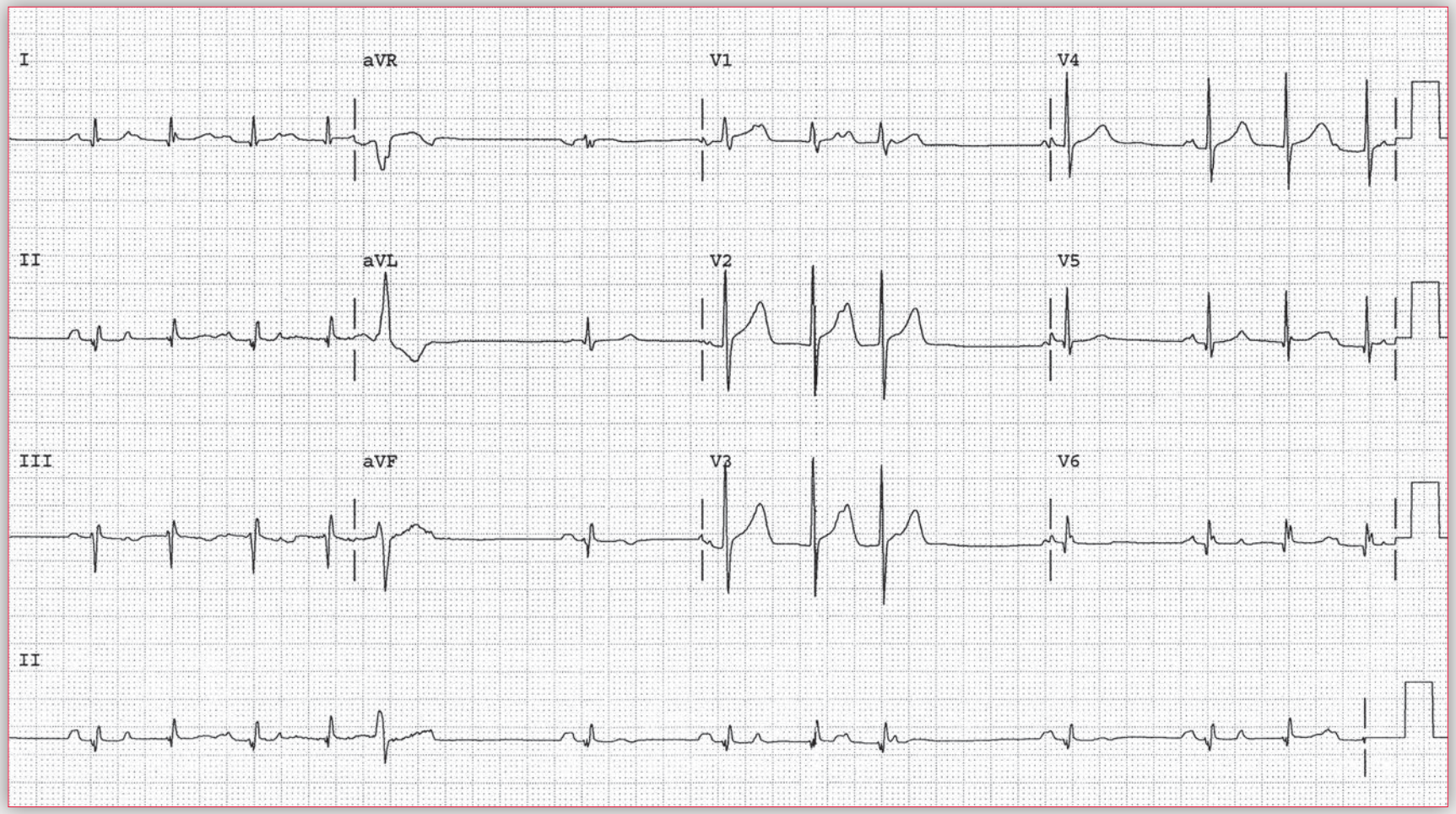
ECG 98C shows a P wave (+) before each QRS complex with a stable PR interval (0.16 sec), identical to the baseline PR interval in **ECG 98B**. The P wave is positive in leads I, II, aVF, and V4-V6. The rate is 60 bpm. Hence this is a normal sinus rhythm. The QRS complex duration, axis, and morphology are normal. The QT/QTc intervals are normal (420/420 msec). There are minor ST-segment changes in leads III and aVF that are consistent with the occurrence of an inferior wall infarction. The absence of changes of a chronic infarct pattern (Q waves and T-wave inversions) suggests that there has not been any significant myocardial damage and that thrombolysis was successful. ■

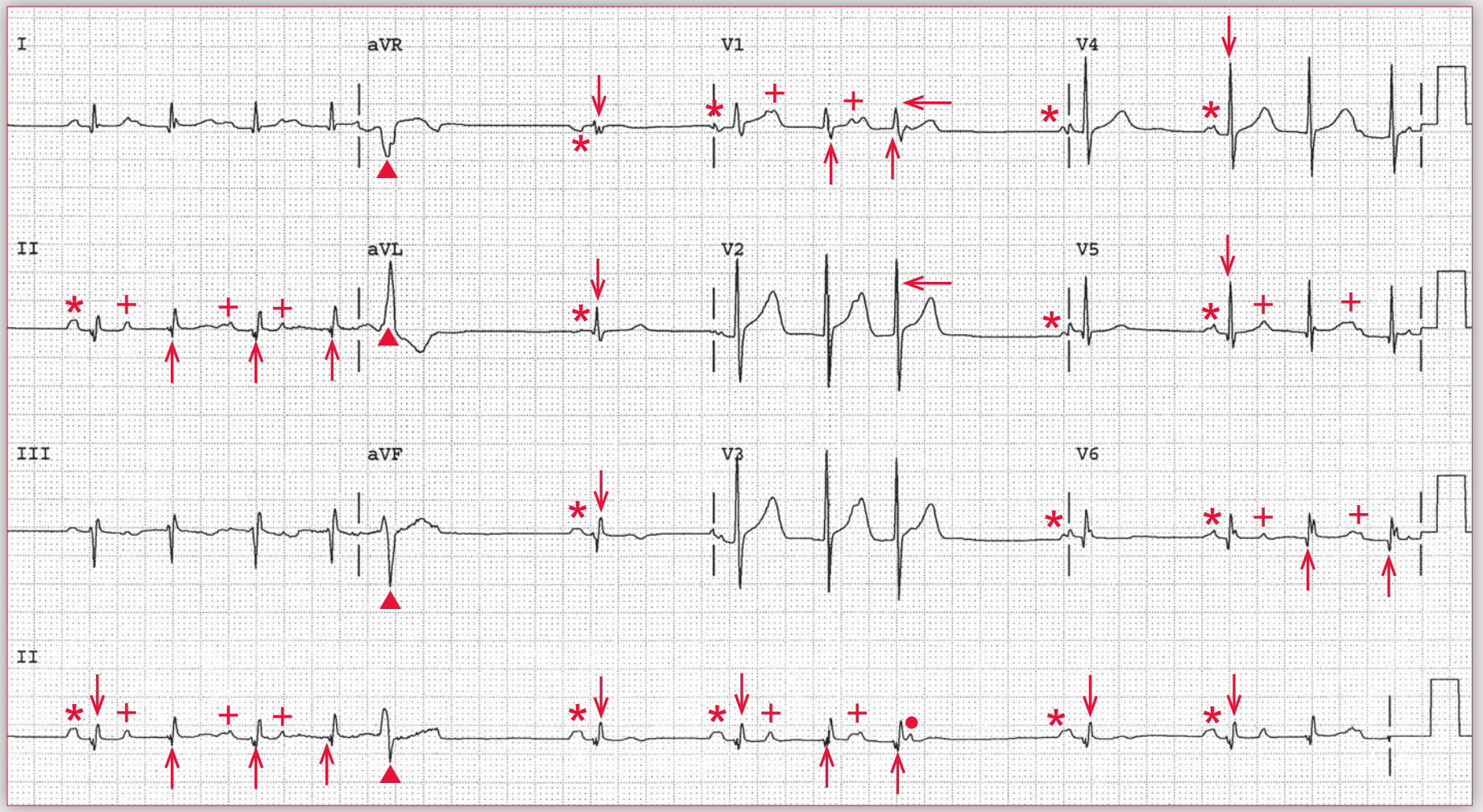
Notes

Practice Case 99

A 48-year-old man presents for a regularly scheduled hemodialysis session. His ECG is shown below.

What is the abnormality?





ECG 99 Analysis: Multifocal atrial premature complexes, counterclockwise rotation (early transition)

The average heart rate is 64 bpm. P waves are most apparent in the lead II rhythm strip. The first, sixth, seventh, 10th, and 11th QRS complexes (↓) are preceded by P waves (*) that have the same morphology and identical PR intervals (0.18 sec). These are sinus P waves and represent the baseline P wave and PR interval. The underlying sinus rate is 60 bpm. After the first QRS complex are three premature QRS complexes (↑) that have the same width and morphology. Each QRS complex is preceded by a premature P wave (+), each of which is different. These are, therefore, multifocal premature atrial complexes (PACs) or a multifocal atrial triplet (*ie*, three sequential PACs). The fifth complex is a premature ventricular complex (▲), after which there are two sinus beats (*). Following these QRS complexes are two sequential PACs (↑) that have different P-wave morphologies (+) (*ie*, a multifocal atrial couplet). A nonconducted P wave (●) can be seen after the second PAC.

The QRS complexes have a normal duration (0.08 sec). The QRS complex morphology is normal, although there is a prominent R wave in lead V1 and a tall R wave in lead V2 (←). This is early transition or counterclockwise rotation in the horizontal axis, which is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, left ventricular forces develop early in the precordial leads.

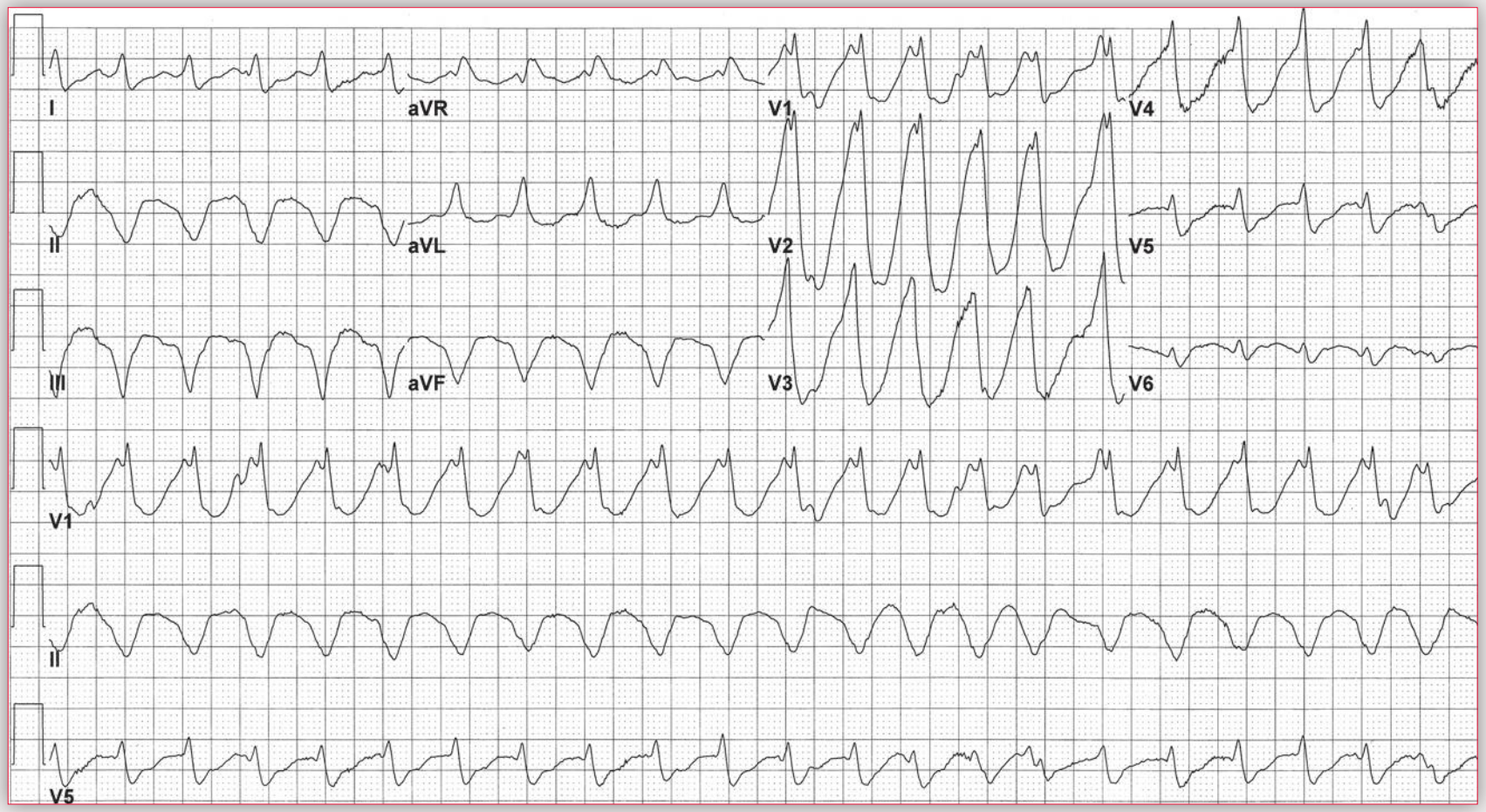
There is low voltage in the limb leads (QRS complex amplitude < 5 mm in each lead). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/360 msec).

PACs are common and may be even more so in patients with renal insufficiency on dialysis in whom electrolyte and pH changes are common. Unifocal PACs are more frequent. Multifocal PACs may be seen more often in patients with electrolyte abnormalities or in those with significant abnormalities of the atrial myocardium. ■

Practice Case 100

A 91-year-old woman presents to the emergency department with worsening symptoms of dyspnea, orthopnea, lightheadedness, and lower extremity edema. On physical examination, jugular venous distention as well as intermittent prominent jugular venous pulsations are noted. Her pulse is regular but tachycardic and her blood pressure is 100/70 mm Hg, although it has some variability while being obtained. Her point of maximum impulse is laterally displaced, and there is an audible but variable S1 and

ECG 100A

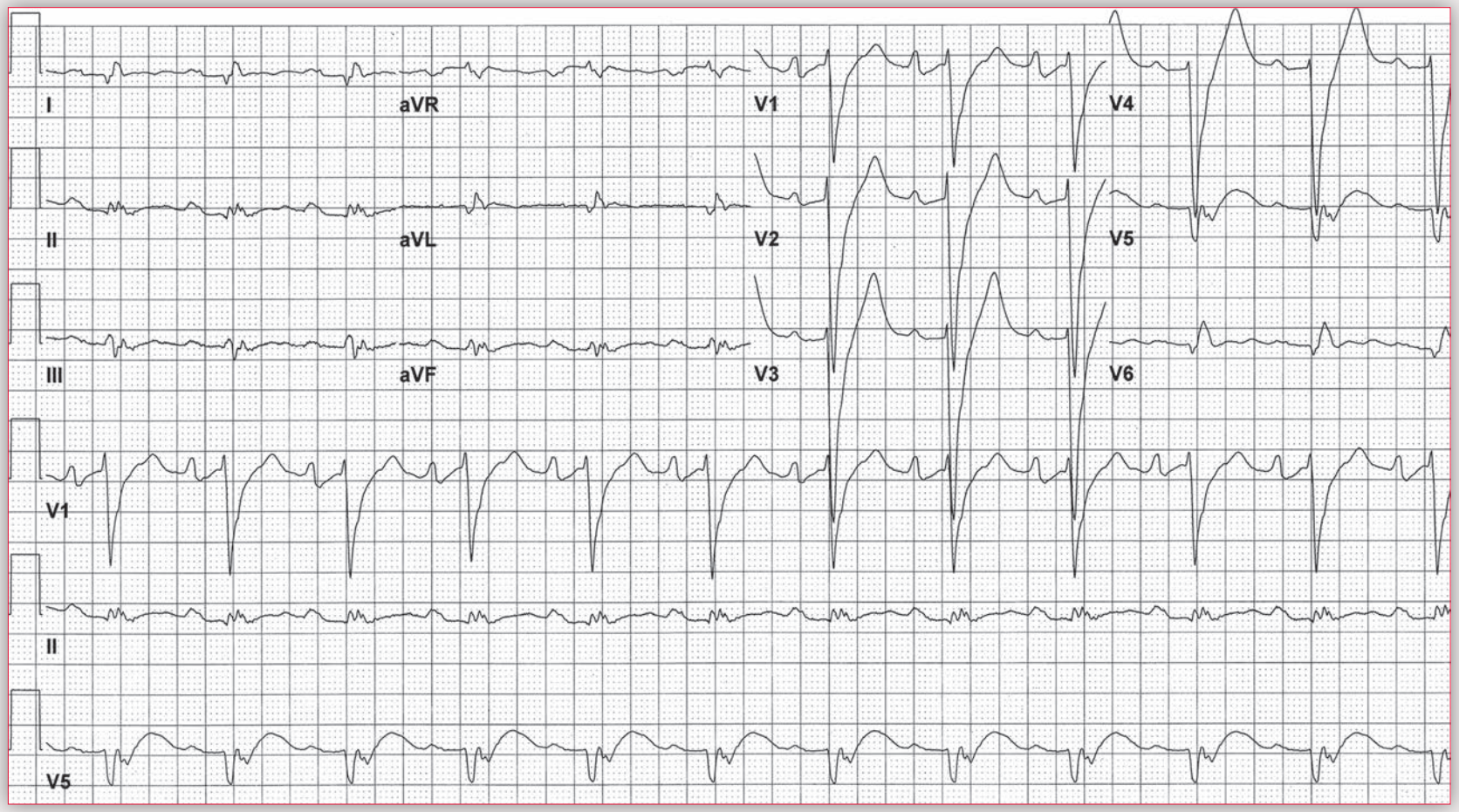


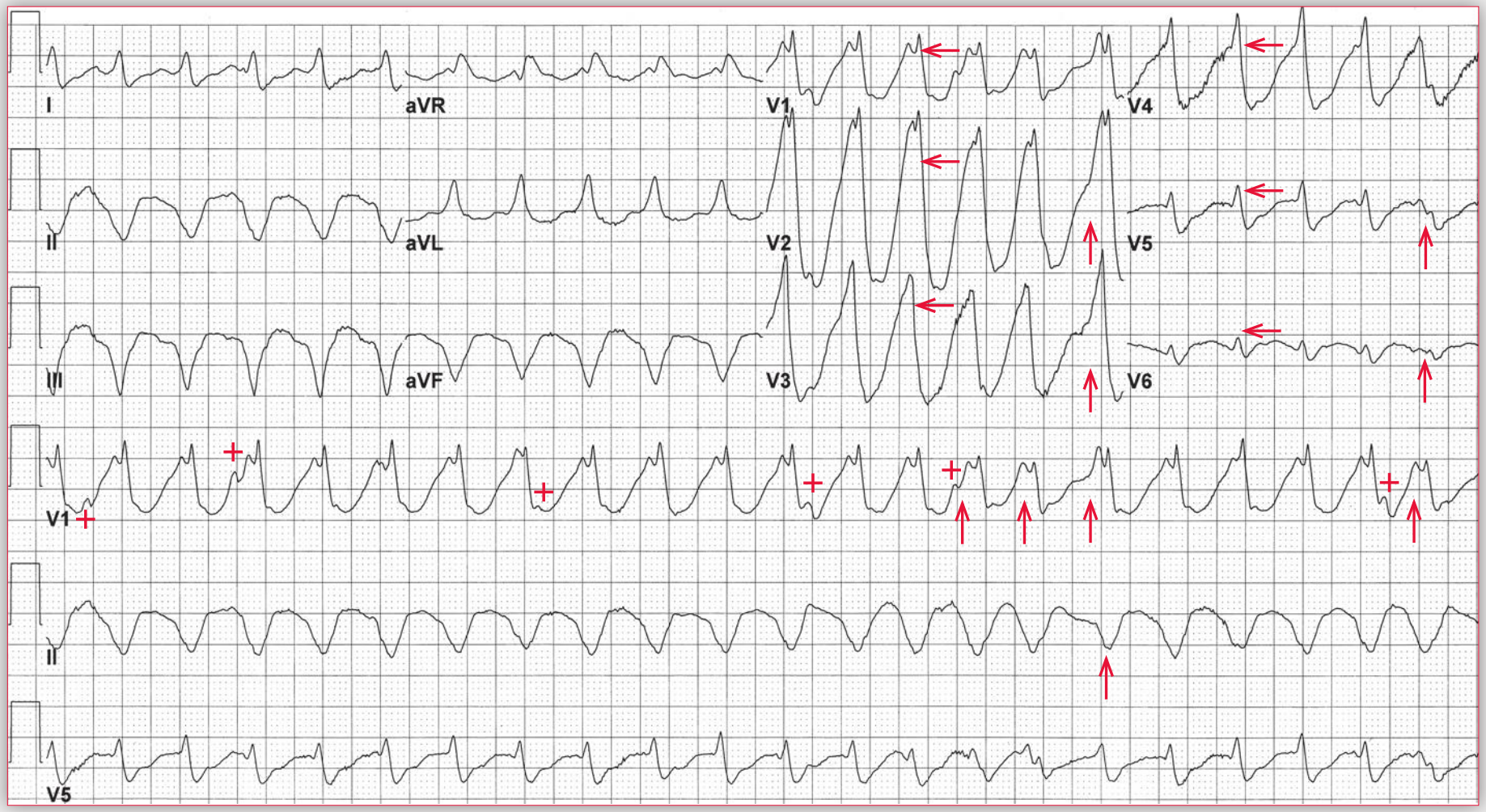
a normal S2. S3 is also audible. Rales are heard throughout the lung fields, and pitting edema is present in the bilateral lower extremities. An ECG is obtained (ECG 100A). The patient's baseline ECG (100B) is shown for comparison.

What is the rhythm abnormality?

Given the patient's baseline ECG (100B), what is the most likely underlying etiology of the arrhythmia?

ECG 100B





ECG 100A Analysis: Monomorphic ventricular tachycardia, AV dissociation

The rhythm in **ECG 100A** is regular at a rate of 130 bpm. The QRS complex duration is increased (0.22 sec) and the axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). Although no distinct atrial activity is seen, some variable waveforms (**+**) are present, best seen in the lead V1 rhythm strip between the 1st and 2nd QRS complexes, before the 4th QRS complex, between the 12th and 13th QRS complexes, between the 14th and 15th QRS complexes, and between the last two QRS complexes. These are superimposed P waves that are dissociated from the QRS complexes. In addition, there are subtle changes in QRS morphology (↑). The morphology of the QRS complexes is not typical for either right or left bundle branch block. In addition, there is positive QRS complex concordance across the precordium, with tall R waves from lead V1 through V5 and possibly V6. These features (AV dissociation, changes in QRS complex and ST-T wave morphology, QRS complex width > 160 msec, positive concordance of QRS complexes across the precordium) support a diagnosis of sustained ventricular tachycardia. The changes in the QRS complex and ST-T wave morphology are due to the fact that ventricular activation is not via the normal His-Purkinje system but rather is by direct myocardial activation. This may result in changes in the sequence or direction of myocardial activation. In contrast, any sinus, atrial or junctional rhythm is conducted to the ventricle through the same pathway (*ie*, AV node His-Purkinje system) and, therefore, all the QRS complexes and ST-T waves are uniform. Positive QRS concordance is also seen only when there is direct myocardial activation, such as with a ventricular complex, a preexcited QRS complex (Wolff-Parkinson-White) or a paced complex. Because all the QRS complexes have the same or similar morphology, this is monomorphic ventricular tachycardia. The QRS complex duration of 0.22 second is wider than is usually seen with ventricular tachycardia and suggests the presence of either an underlying severe cardiomyopathy (with diffuse fibrosis) or hyperkalemia.

The physical examination of this patient also suggests ventricular tachycardia as the diagnosis. The intermittent and prominent jugular venous pulsations represent cannon A waves resulting from AV dissociation and intermittent right atrial contraction against a closed tricuspid valve. AV dissociation also leads to a variable S1 as a result of a wide range of tricuspid and mitral valve leaflet excursion at the time of ventricular systole. In addition, there is variability of blood pressure, which also reflects AV dissociation with variable filling of the left ventricle and changes in stroke volume due to variability in the relationship between atrial and ventricular contraction. This patient is clearly in significant heart failure, although it is not certain whether ventricular tachycardia precipitated the heart failure or heart failure precipitated ventricular tachycardia.

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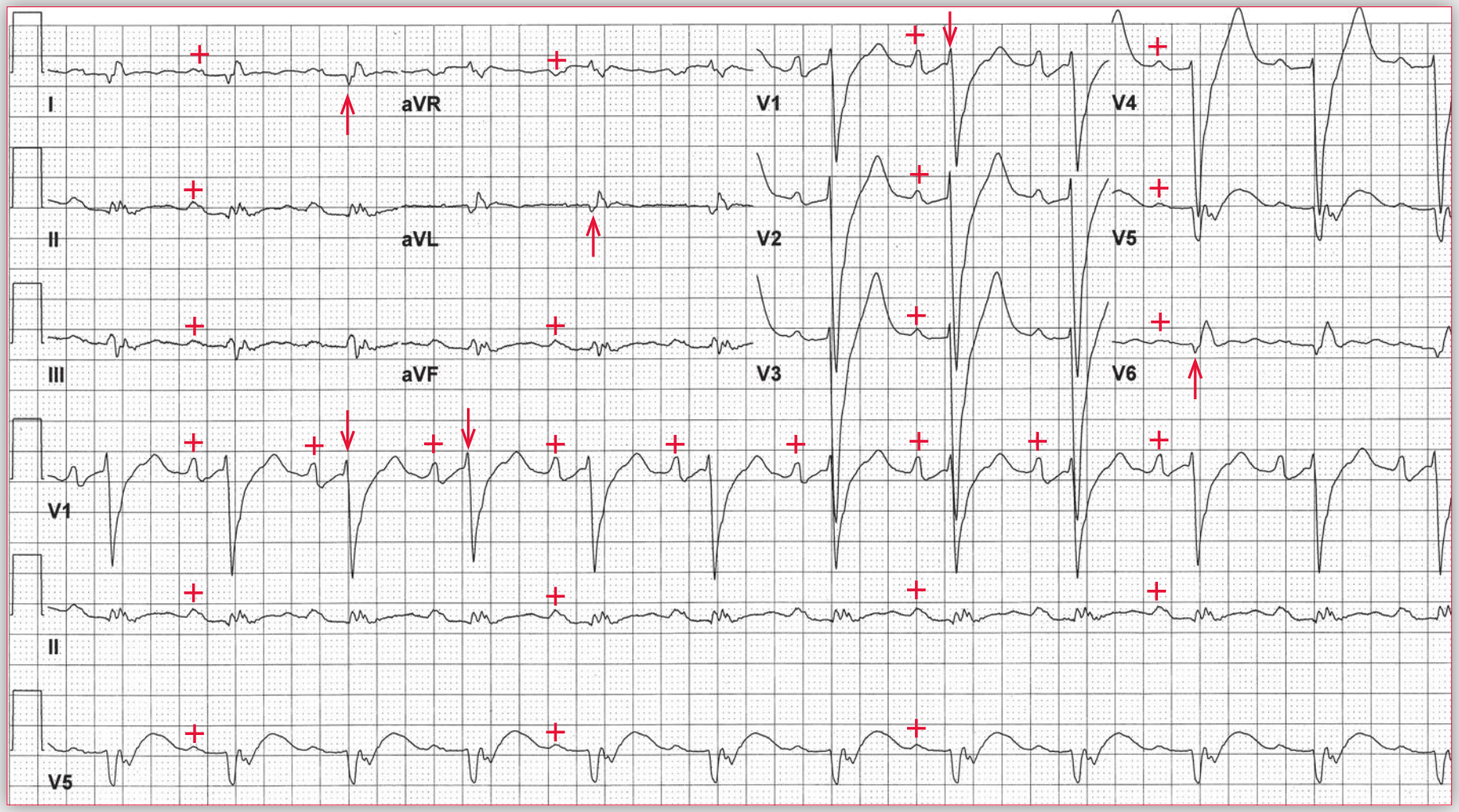


ECG 100B Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), right atrial hypertrophy, intraventricular conduction delay, old anterolateral and lateral myocardial infarction

In **ECG 100B** there is a regular rhythm at a rate of 70 bpm. P waves (**+**) can be seen before each QRS complex with a stable PR interval of 0.28 second. The P waves are positive in leads I, II, aVF, and V4-V6; hence this is a sinus rhythm with first-degree AV block or prolonged AV conduction). The QRS complex duration is prolonged (0.18 sec), and there is a morphology that resembles a left bundle branch block (LBBB) with a deep S wave in lead V1 and a broad R wave in leads I and V5-V6. However, there are septal forces seen, *ie*, a prominent R wave in lead V1 (↓) and Q waves in leads I, aVL, and V6 (↑). Importantly, septal forces are not seen with an LBBB because the septal or median fascicle, which innervates the intraventricular septum, is a branch of the left bundle. The septum, which is the first part of the ventricle to be activated, is depolarized in a left-to-right direction, accounting for the septal R wave in lead V1 and septal Q waves in leads I, aVL and V6. Hence this is a nonspecific intraventricular conduction delay (IVCD). Importantly, an IVCD is due to slowing of conduction through the normal His-Purkinje system; hence abnormalities of the left ventricle can be diagnosed. In contrast, with an LBBB activation is not via the normal His-Purkinje system but is by direct myocardial activation. Therefore, abnormalities of the left ventricle cannot be interpreted reliably. The QT/QTc intervals are prolonged (500/540 msec) but are normal when corrected for the prolonged QRS complex duration (400/430 msec).

When the QRS complex is this wide it is likely that the IVCD is the result of dilated cardiomyopathy with significant fibrosis causing marked impulse conduction slowing. In this case there are significant, broad Q waves in leads I, aVL and V6 that are due to an old lateral and anterolateral myocardial infarction, the result of coronary disease. This therefore is an ischemic cardiomyopathy. Although these Q waves are wide and not septal, they have the same meaning of septal Q waves, representing forces going in a left-to-right direction as a result of myocardical infarction. Left-to-right forces are not seen in an LBBB as all forces are directed from right to left. Importantly, the Q waves present with a chronic myocardial infarction are not usually seen with an LBBB. The QRS complexes during sinus rhythm are very different from those seen during ventricular tachycardia in **ECG 100A**. The very wide QRS complexes seen during the ventricular tachycardia are probably due to the underlying cardiomyopathy and fibrosis with very slow intraventricular conduction.

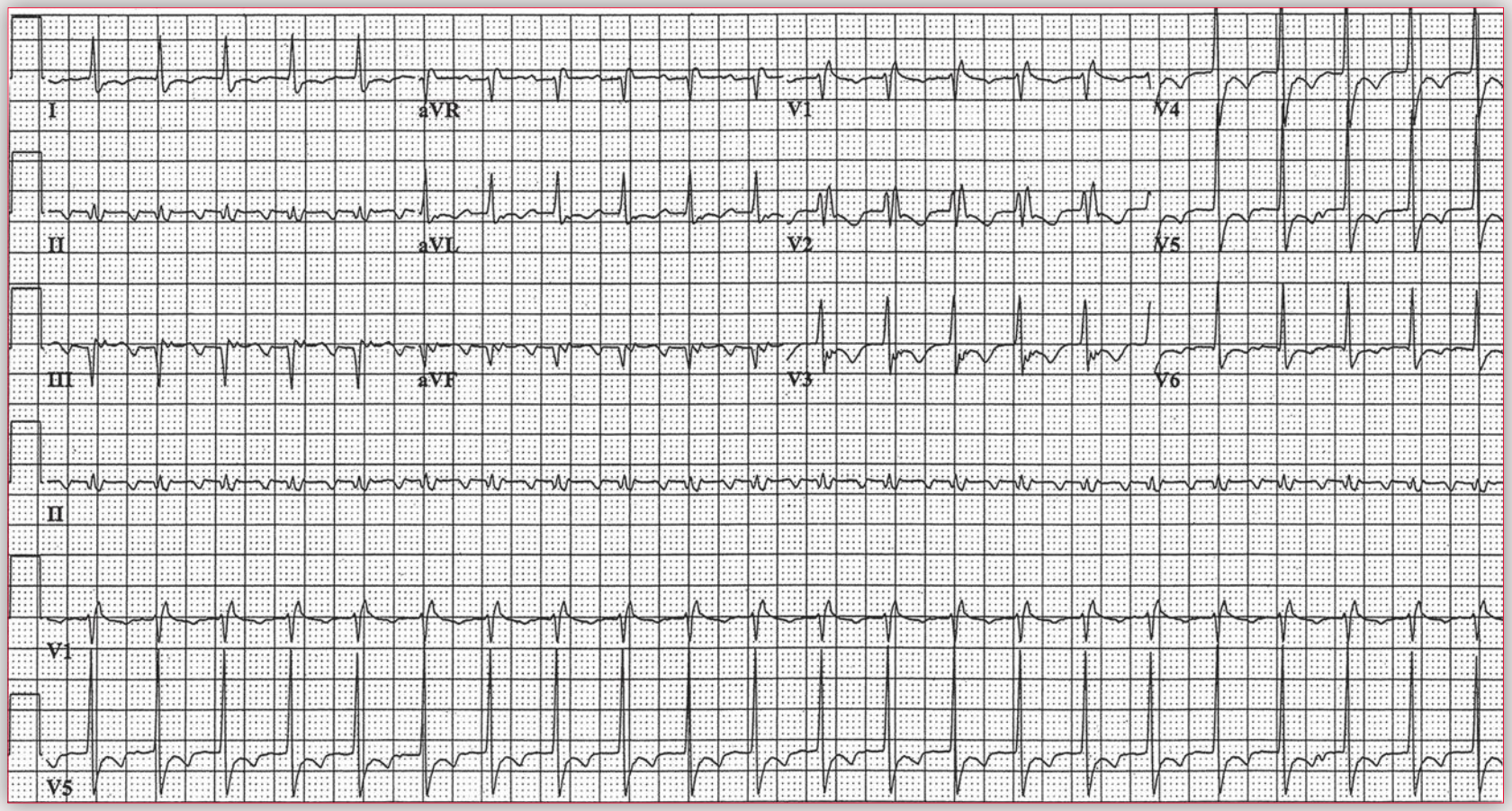
The P waves are tall and peaked in lead V1, suggesting right atrial hypertrophy. ■

Practice Case 101

A 14-year-old girl with known unrepaired ventricular septal defect is flown to the United States for evaluation and treatment. She is brought straight to the emergency department because she appears unwell to the airline's flight staff.

On presentation, the patient appears cyanotic and an interpreter notes that she experienced sudden-onset dyspnea and palpitations a few hours before the end of the transatlantic journey. Her oxygen saturation is 89% on ambient air, pulse 140 bpm, and

ECG 101A



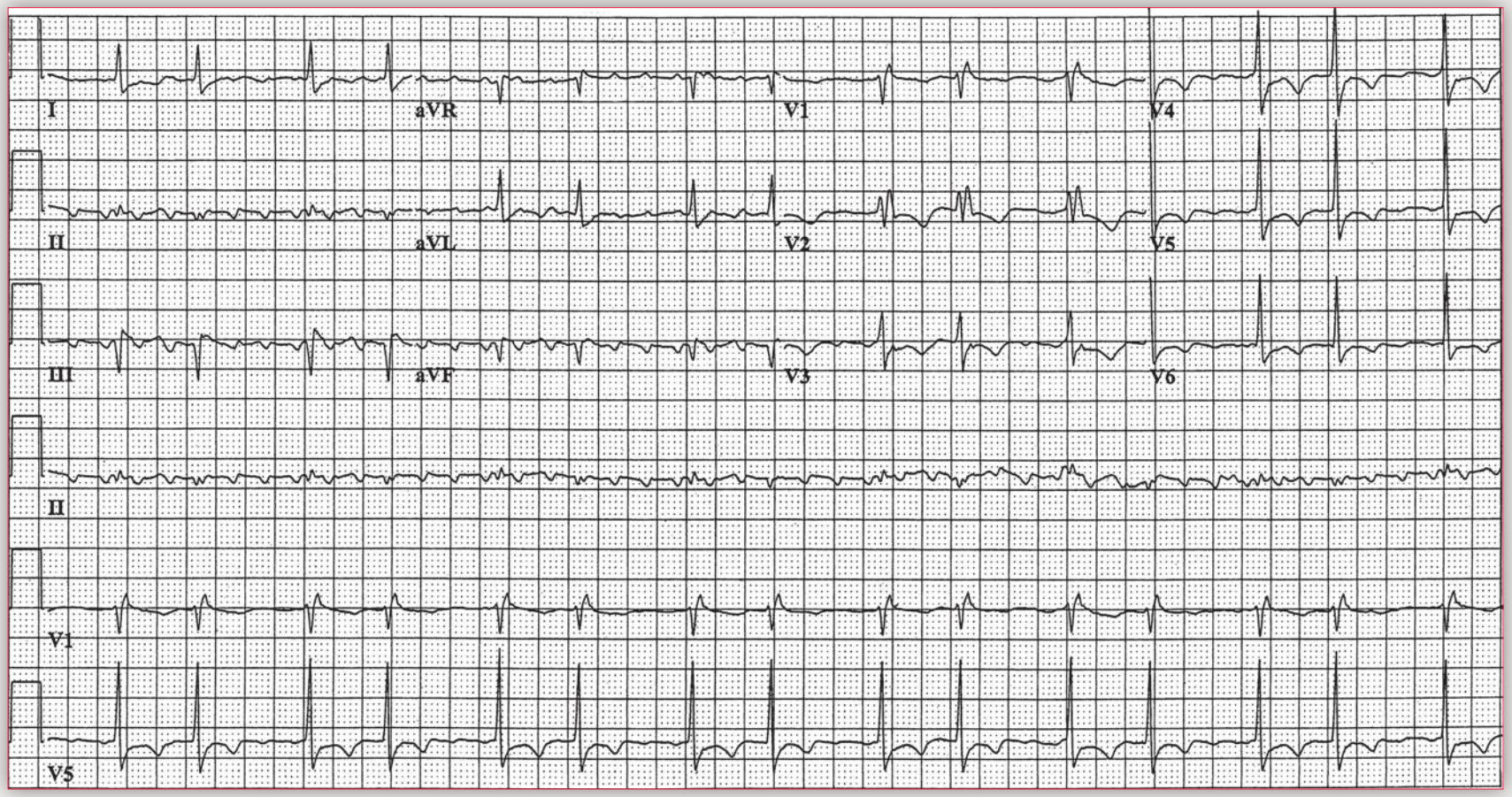
Practice Case 101

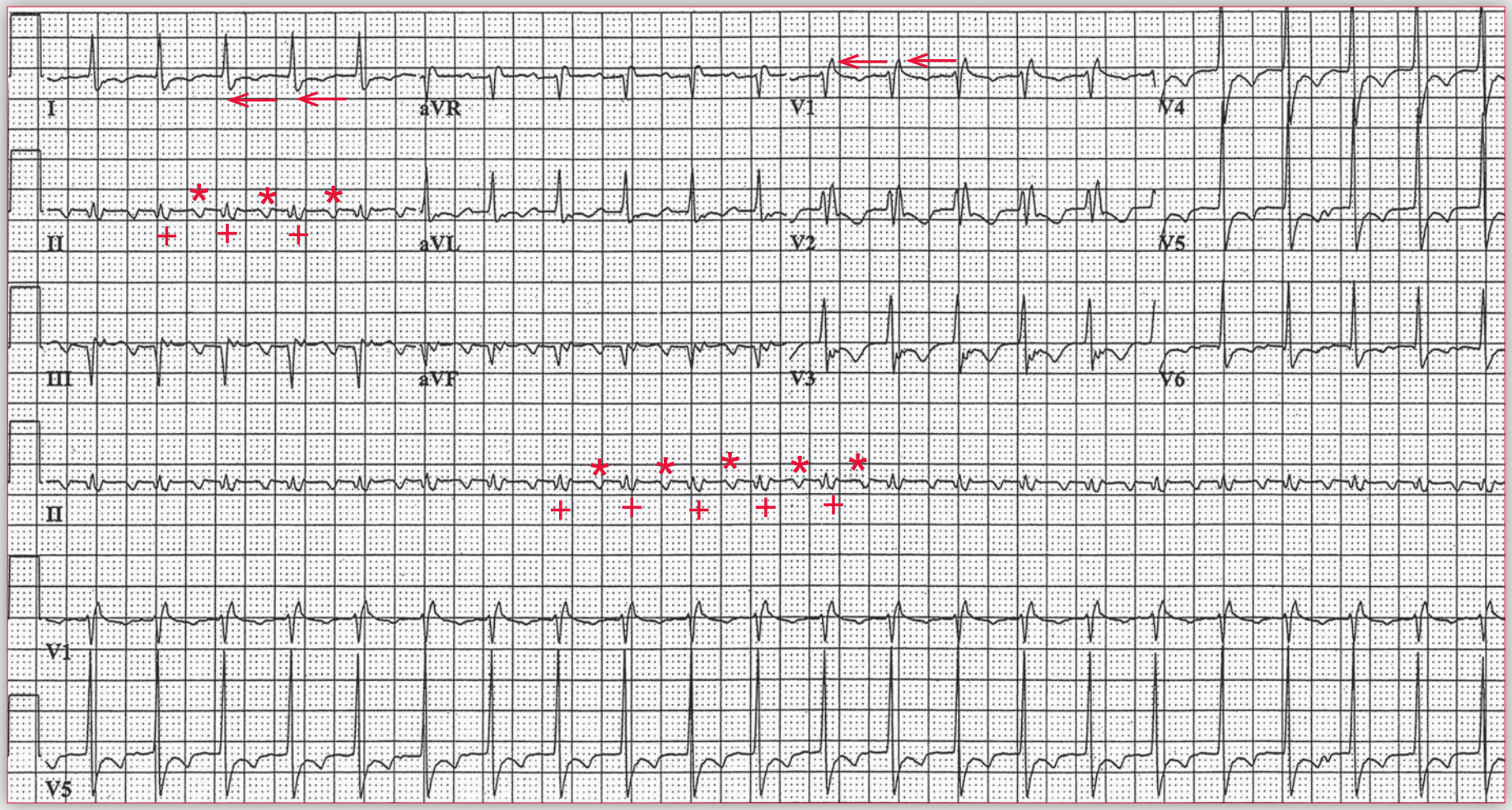
blood pressure 85/40 mm Hg. Jugular venous pressure is 14 cm H_2O with large, rapid A waves, a blunted X descent, and a large CV wave. Cardiac exam is notable for a right parasternal lift and a harsh pansystolic murmur at the lower sternal border. Lungs are clear; there is an enlarged, tender, pulsatile liver and somewhat asymmetric lower extremity edema.

Two ECGs are obtained. The first (ECG 101A) represents the patient's index tracing, and the second (ECG 101B) was obtained during carotid sinus massage.

What is the underlying rhythm?

ECG 101B





ECG 101A Analysis: Atrial flutter with 2:1 conduction, right bundle branch block, left axis

In **ECG 101A** there is a regular rhythm at a rate of 140 bpm. The QRS complex duration is increased (0.12 sec). There is an RSR′ morphology in lead V1 (←) and an S wave in leads I and V4-V6 (←), diagnostic for a right bundle branch block (RBBB). There is a physiologic left axis, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). As measured, the QT/QTc intervals are prolonged (320/490 msec). However, when considering the prolonged QRS complex duration, the QT/QTc intervals are normal (280/430 msec).

Negative atrial waveforms can be seen in leads II and aVF (*). A second atrial waveform (+) is seen at the end of the QRS complex, particularly in lead II. Although it looks like an S wave, it has the same morphology as the atrial waveform that is seen before the QRS complex and the interval between the two atrial waveforms is the same and stable, at a rate of 280 bpm. The regular atrial rate of 280 bpm is diagnostic for atrial flutter, and there is 2:1 AV conduction.

Atrial flutter is often a difficult rhythm to diagnose because one of the two flutter waves may be within the QRS complex, at the end of the QRS complex (resembling an S wave or even ST-segment depression), or at the beginning of the QRS complex (suggesting a Q wave).

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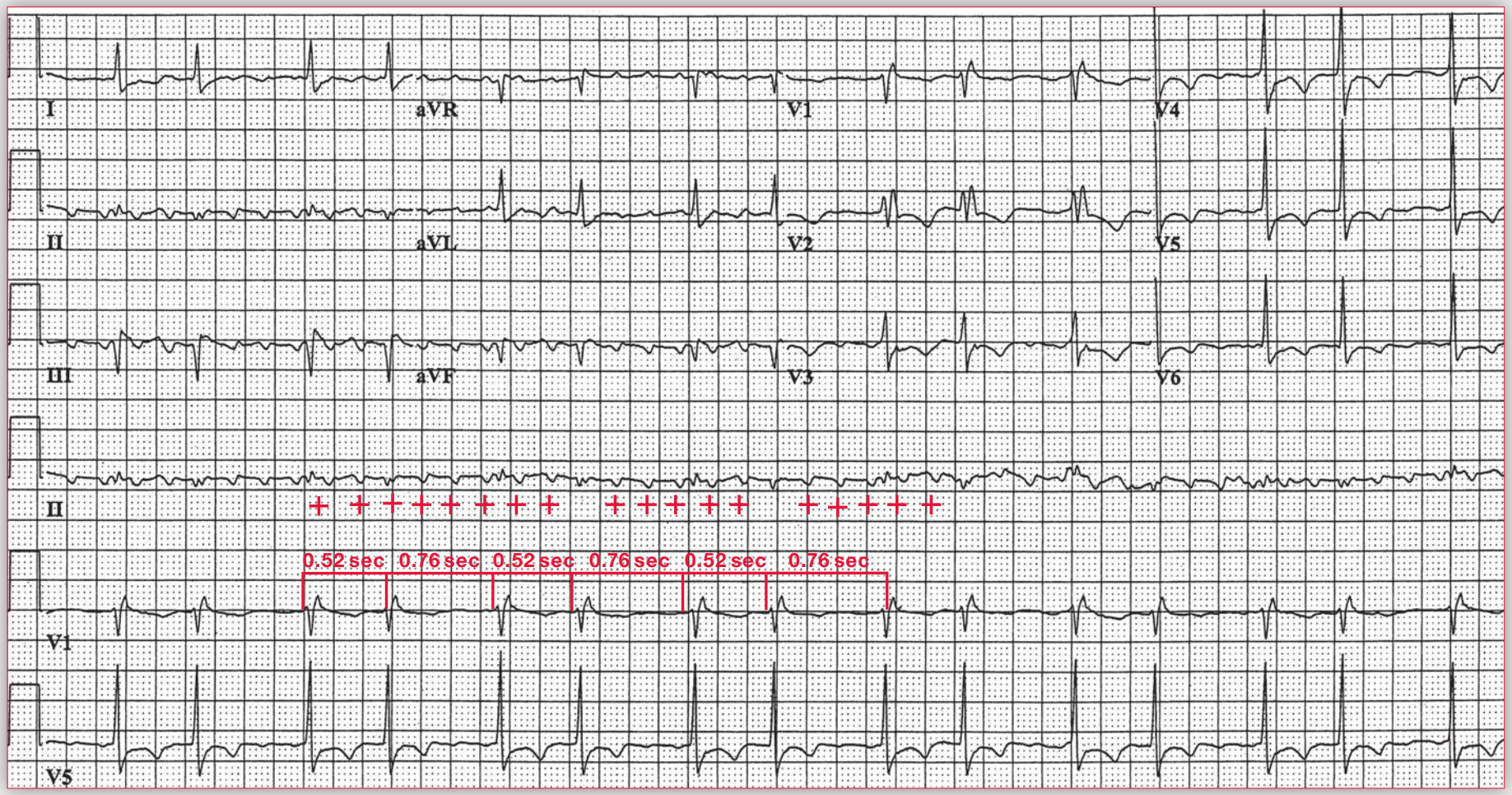


ECG 101B Analysis: Atrial flutter with variable conduction (2:1 and 4:1)

In **ECG 101B**, the QRS complex duration, morphology, and axis are the same as in **ECG 101A**. The rhythm is irregular, but there is a pattern with all of the short and long RR intervals being the same. Hence the rhythm is regularly irregular. There are now distinct atrial waveforms noted (**+**) as a result of increased AV nodal block. The atrial rate is regular at 280 bpm (identical to the atrial rate in **ECG 101A**), and the atrial waveforms are negative in leads II and aVF. Hence this is atrial flutter and there is variable AV conduction (2:1 alternating with 4:1). The variable AV conduction is the result of carotid sinus pressure that increased vagal inputs to the AV node, slowing conduction through this structure.

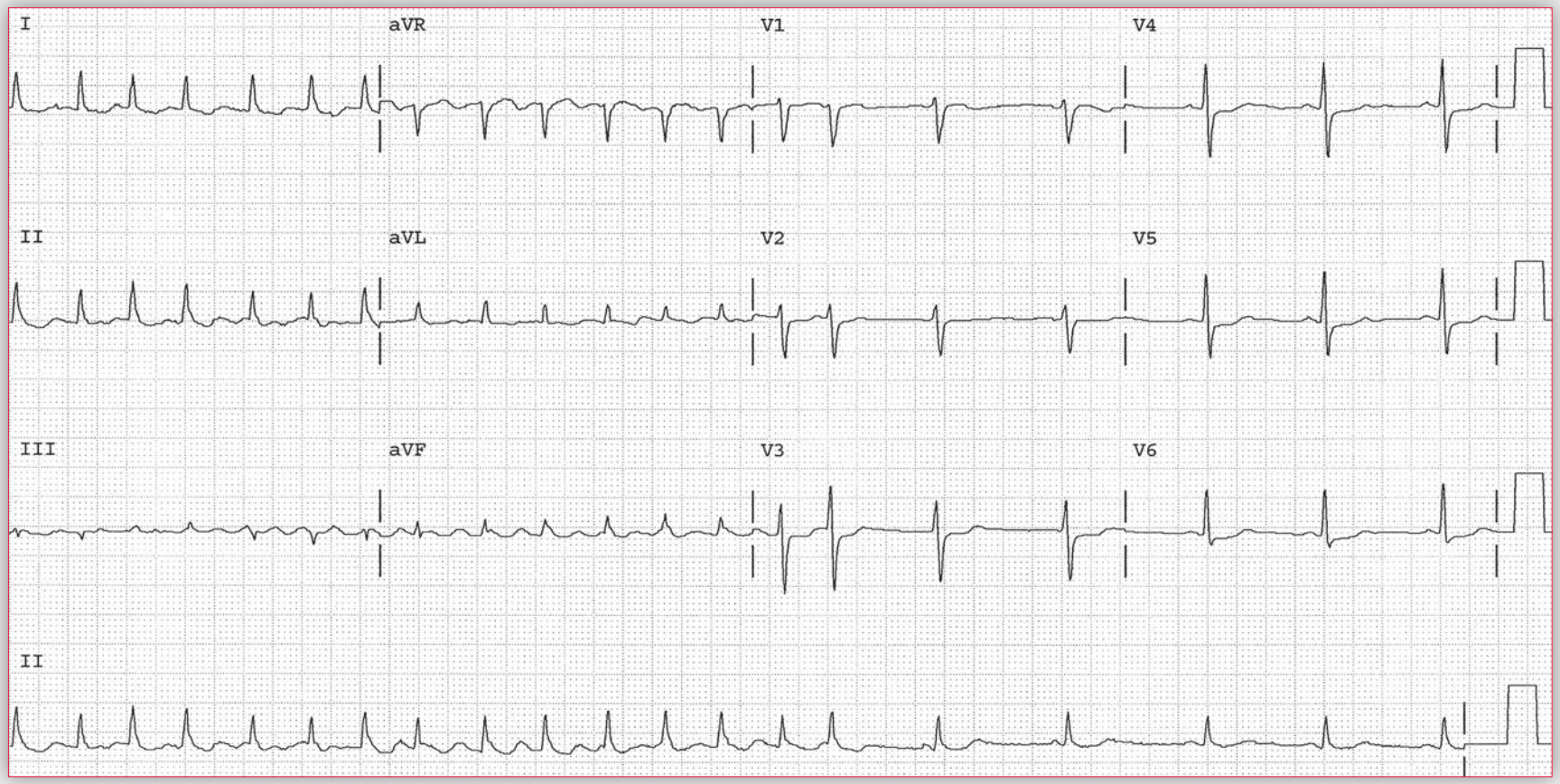
Atrial arrhythmias are common in patients with congenital heart disease, especially when associated with left-to-right shunting. This results in the development of right ventricular and right atrial hypertrophy. The acute onset of atrial flutter, with the rapid atrial and ventricular rate, will result in significant hemodynamic impairment in such patients and is the likely cause for the sudden clinical deterioration in this patient. ■

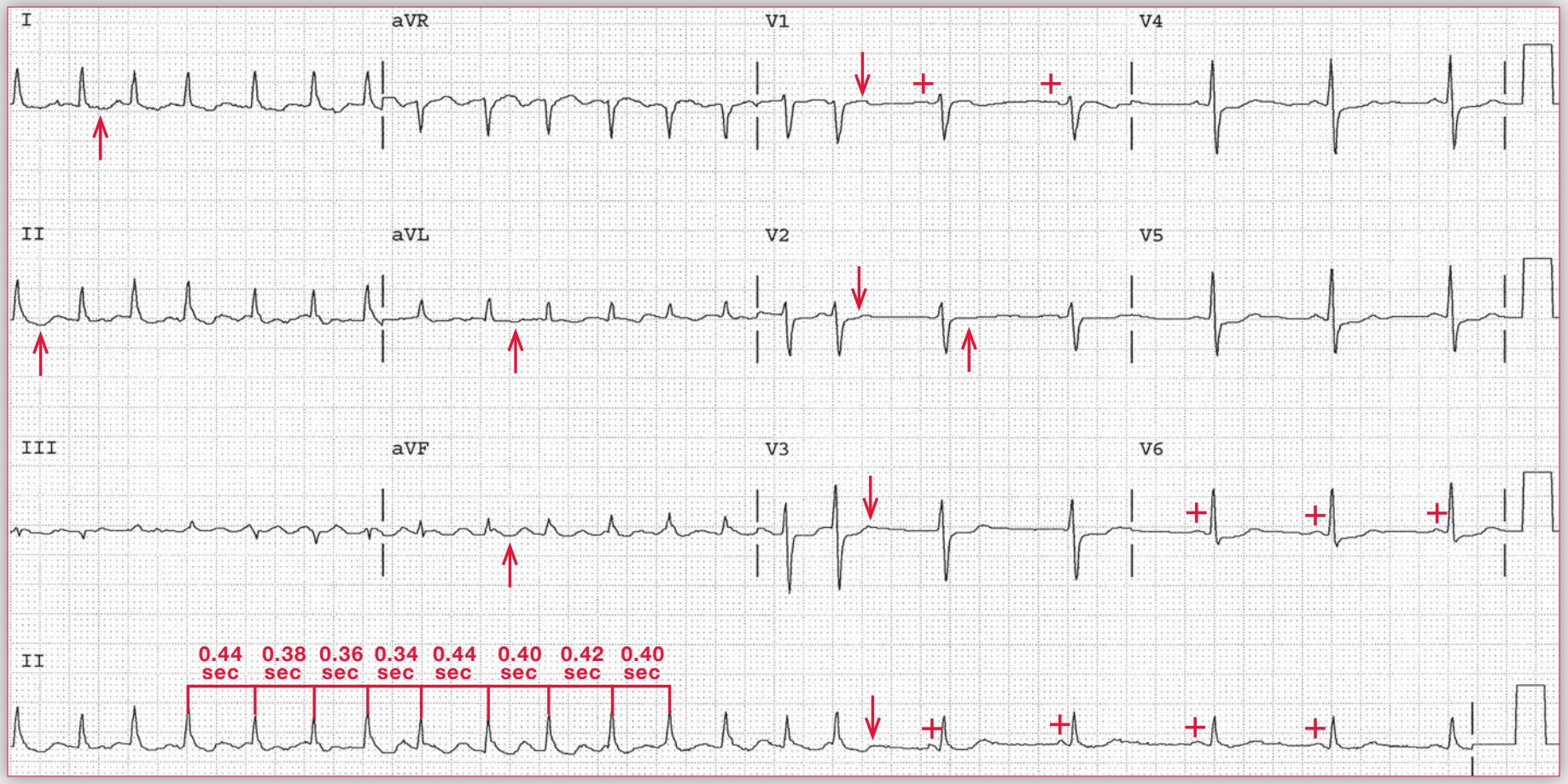
Notes

Practice Case 102

A medical student approaches you with a puzzling ECG. As he hands you the ECG, he asks why the patient's rhythm suddenly slowed.

What is the explanation for the change in RR interval that the medical student has noticed?





ECG 102 Analysis: Atrial fibrillation with conversion to normal sinus rhythm, low-voltage limb leads, nonspecific ST-segment abnormalities

The first part of the ECG shows a rhythm that is irregularly irregular at a rate of 156 bpm. There are three supraventricular rhythms that are irregularly irregular: (1) sinus arrhythmia, in which there is one P-wave morphology and PR interval; (2) multifocal atrial rhythm (wandering atrial pacemaker) with a rate less than 100 bpm or multifocal atrial tachycardia with rate greater than 100 bpm in which there are three or more different P-wave morphologies and no P-wave morphology is dominant, or (3) atrial fibrillation, in which there is no organized atrial activity but there are fibrillatory waves. There is no obvious atrial activity, and hence the rhythm is atrial fibrillation with a rapid ventricular response. However, the rapid and irregular arrhythmia terminates abruptly (↓) to a slower and regular rhythm (rate, 76 bpm) with P waves (+) that are present before each QRS complex with a stable PR interval (0.16 sec). This is, therefore, a normal sinus rhythm. The QRS complex morphology, axis, and duration are the same during atrial fibrillation and sinus rhythm. The QRS complex duration (0.08 sec) and morphology are normal; and the axis is normal, between 0° and 90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/430 msec). There is low voltage in the limb leads (QRS complex amplitude < 5 mm in each limb lead). There are also diffuse nonspecific ST-T wave abnormalities (↑).

The spontaneous reversion of atrial fibrillation to normal sinus rhythm means that the atrial fibrillation is paroxysmal or intermittent. This is a frequent occurrence in patients with new-onset atrial fibrillation. Often the patient is unaware of the paroxysms of atrial fibrillation as they may be brief. Intermittent atrial fibrillation does not necessitate therapy unless the patient is symptomatic during the arrhythmia. In this situation, the use of a β-blocker or calcium-channel blocker to control the rapid ventricular rate may be beneficial. Suppression of the arrhythmia with an anti-arrhythmic drug is not always necessary unless the episodes are frequent, long lasting, and symptomatic. In this situation a "pill in the pocket" or "cocktail drug therapy" approach would be useful. This approach involves the use of a large dose of a class IA or IC anti-arrhythmic drug to be taken at the onset of atrial fibrillation. The drug should be evaluated for efficacy and safety before using this approach. In general, one-half of the usual daily dose of the agent is given as a single dose. This will establish a therapeutic blood level within 2 to 3 hours, at which time the arrhythmia will terminate. If this approach is successful, it avoids the need for long-term drug therapy for an arrhythmia that is episodic.

For patients who do not have any symptoms from the arrhythmia, a pill in the pocket approach is not indicated because the arrhythmia is not recognized when it occurs. No therapy may be needed for such a patient.

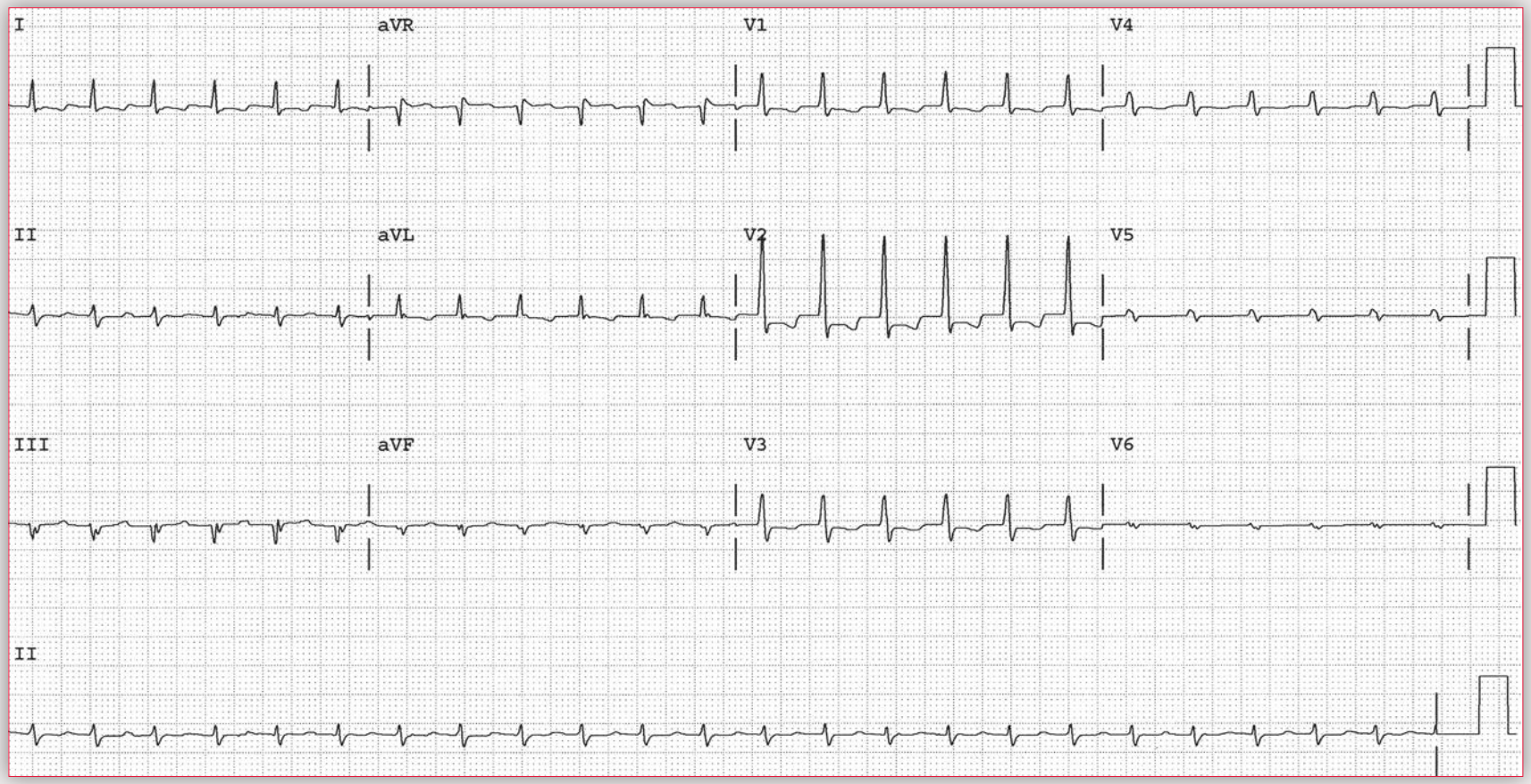
One important consideration is the need for anticoagulation in these patients. Although no trials specifically address this issue in patients with intermittent atrial fibrillation, these patients are believed to have the same embolic risk as those with chronic atrial fibrillation and hence anticoagulation therapy should be considered based on the CHADS2 score. Although data are lacking, things to consider with regard to anticoagulation are the frequency and duration of the episodes of atrial fibrillation. Importantly, a discussion with the patient is required for his or her input into any decision about anticoagulation. ■

Practice Case 103

A 75-year-old woman presents to the emergency department following 2 hours of chest discomfort. She states that the central chest pressure began suddenly while she was watching TV after dinner. She thought it may have been "heartburn" and so took an oral over-the-counter antacid, which did not provide relief. The discomfort became worrisome enough that she sought medical attention.

She denies a history of coronary disease or ever having had symptoms such as this before. She has a history of diabetes mellitus and dyslipidemia for which she takes rosiglitazone and simvastatin, respectively.

ECG 103A



Practice Case 103

She has chronic arthritis for which she takes an NSAID daily.

Her heart rate is 140 bpm. A quick physical exam reveals no notable findings. An ECG (103A) is obtained. After the physician performs a diagnostic maneuver, a second ECG is obtained (ECG 103B).

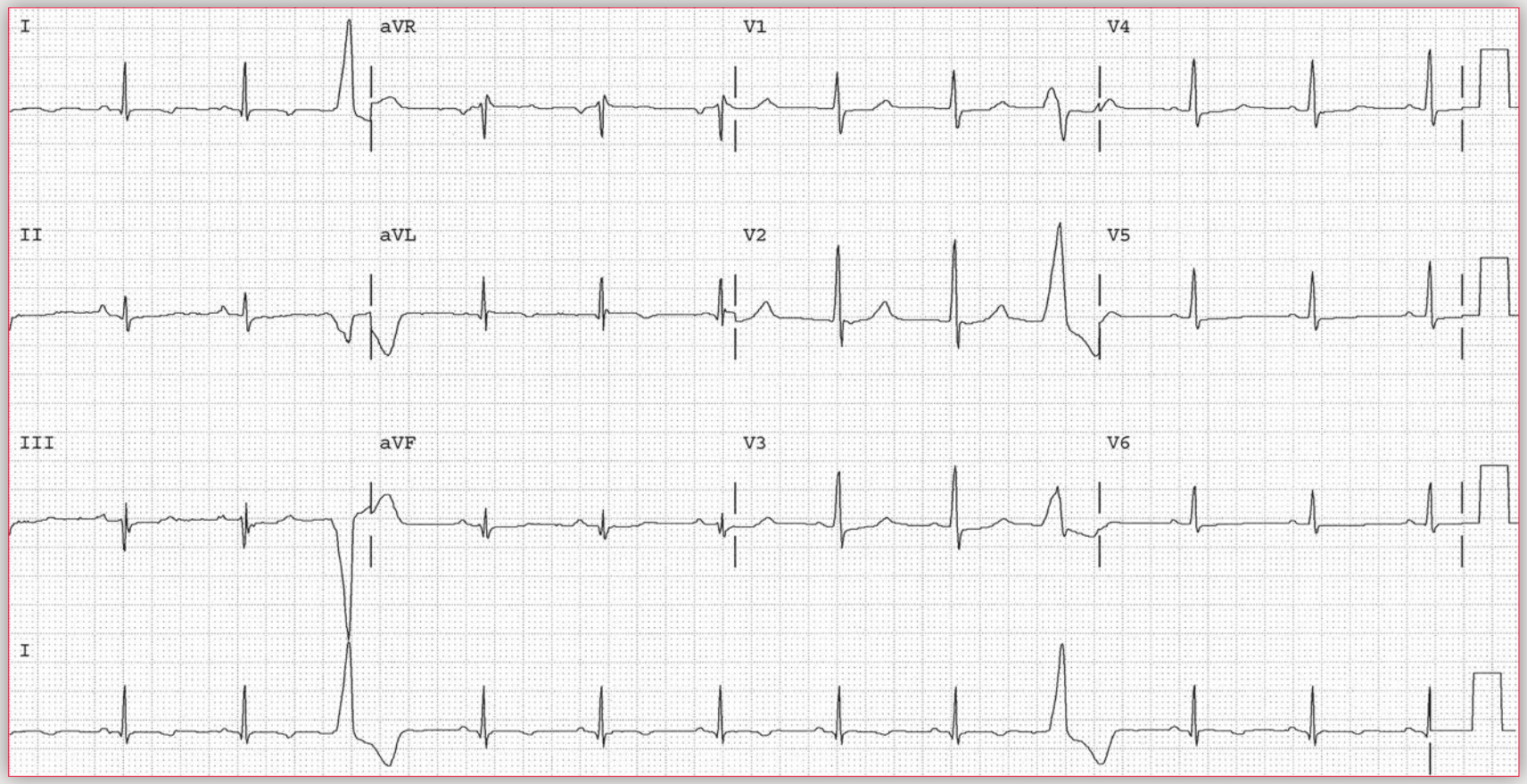
What abnormalities are notable in ECG 103A?

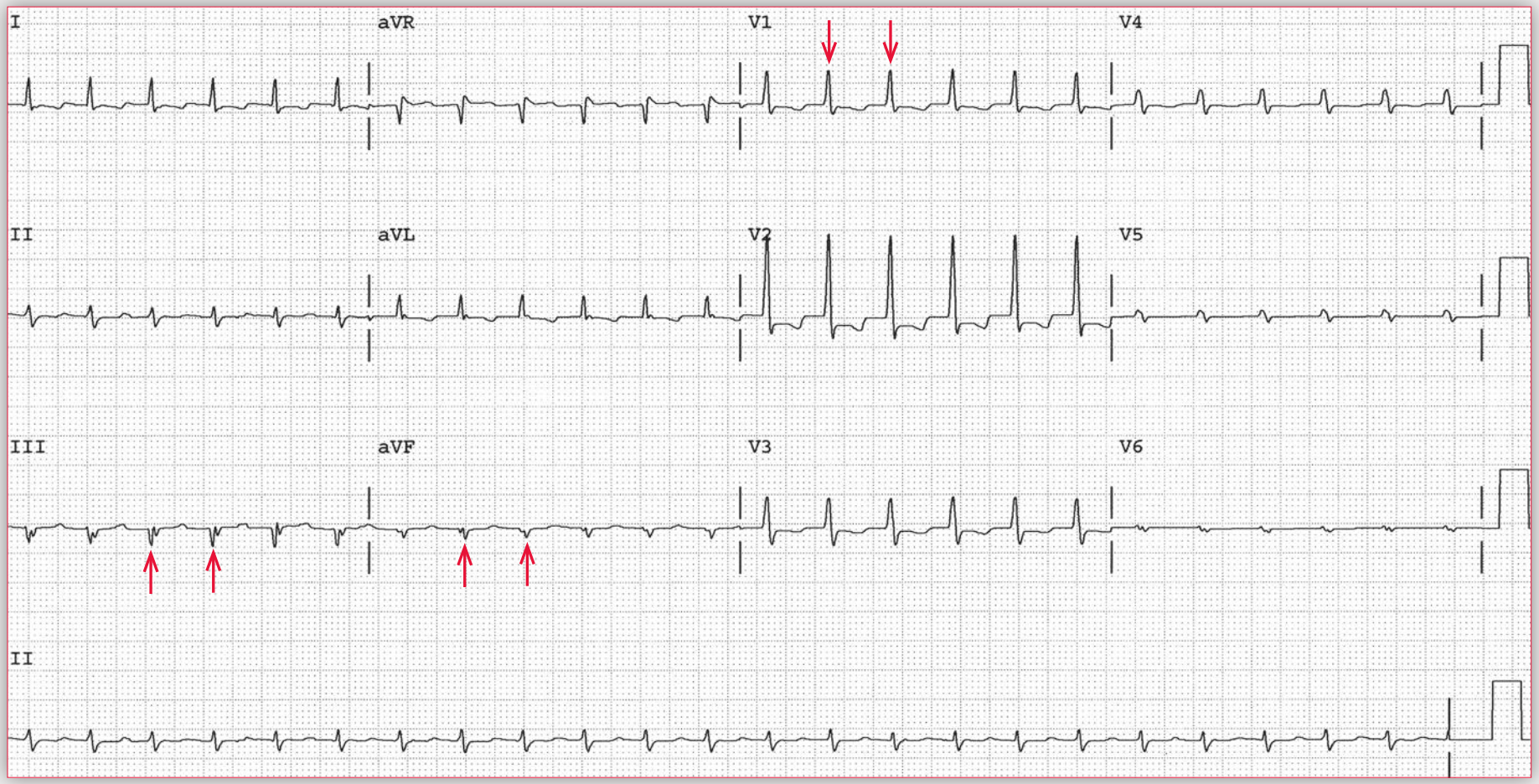
What is the likely diagnosis?

What maneuver might the physician have performed?

Based on the follow-up ECG (103B), what is the diagnosis?

ECG 103B





ECG 103A Analysis: Narrow complex, atrioventricular nodal reentrant tachycardia (AVNRT), left axis, inferoposterior wall myocardial infarction, ST-T wave abnormalities consistent with myocardial ischemia

ECG 103A shows a regular rhythm at a rate of 140 bpm. The QRS complex duration is normal (0.08 sec), as are the QT/QTc intervals (280/430 msec). The axis is leftward (positive QRS complex in leads I and II and negative QRS complex in lead aVF), but the negative QRS complex in lead aVF is the result of a Q wave (↑). There is also a Q wave in lead III (↑↑). Q waves in leads II and III are diagnostic for an inferior wall myocardial infarction. In addition, there is a tall R wave in lead V1 (↓) (R/S ratio > 1 or R-wave amplitude > 7 mm); along with an inferior wall myocardial infarction, this is most likely the result of posterior wall involvement. However, other causes for a tall R wave in lead V1 include the following:

- Right ventricular hypertrophy (usually associated with a right axis and right atrial hypertrophy)
- Wolff-Parkinson-White pattern (short PR interval and widened QRS complex due to a delta wave)
- Hypertrophic cardiomyopathy (septal hypertrophy with prominent septal Q waves) and usually left ventricular hypertrophy
- Early transition (counterclockwise rotation)
- Duchenne muscular dystrophy (associated with a posterolateral infarct pattern)
- Dextrocardia (reverse R-wave progression in leads V1-V6, right axis, negative P and T waves in leads I and aVL and positive P and T waves in lead aVR)
- Lead switch (leads V1, V2, V3)
- Right-sided leads with reverse R-wave progression
- Normal variant

Also noted are ST-segment depressions, especially in lead V2, along with T-wave abnormalities. These findings are consistent with subendocardial ischemia. There are no P waves before or after the QRS complexes. Hence this is no-RP junctional tachycardia. The most common etiology for this is atrioventricular nodal reentrant tachycardia (AVNRT).

Based on the likely diagnosis of AVNRT, maneuvers to alter the conduction through one part of the re-entrant circuit may be both diagnostic and therapeutic. Physical maneuvers such as carotid sinus massage, Valsalva, ocular pressure, and cold water facial submersion (to elicit the "diving reflex") increase vagal efferent impulses to the AV node and may slow nodal conduction enough (or completely block it) to interrupt the circuit. Pharmacologic therapy with agents such as adenosine, verapamil, or a β-blocker may also have the same effect.

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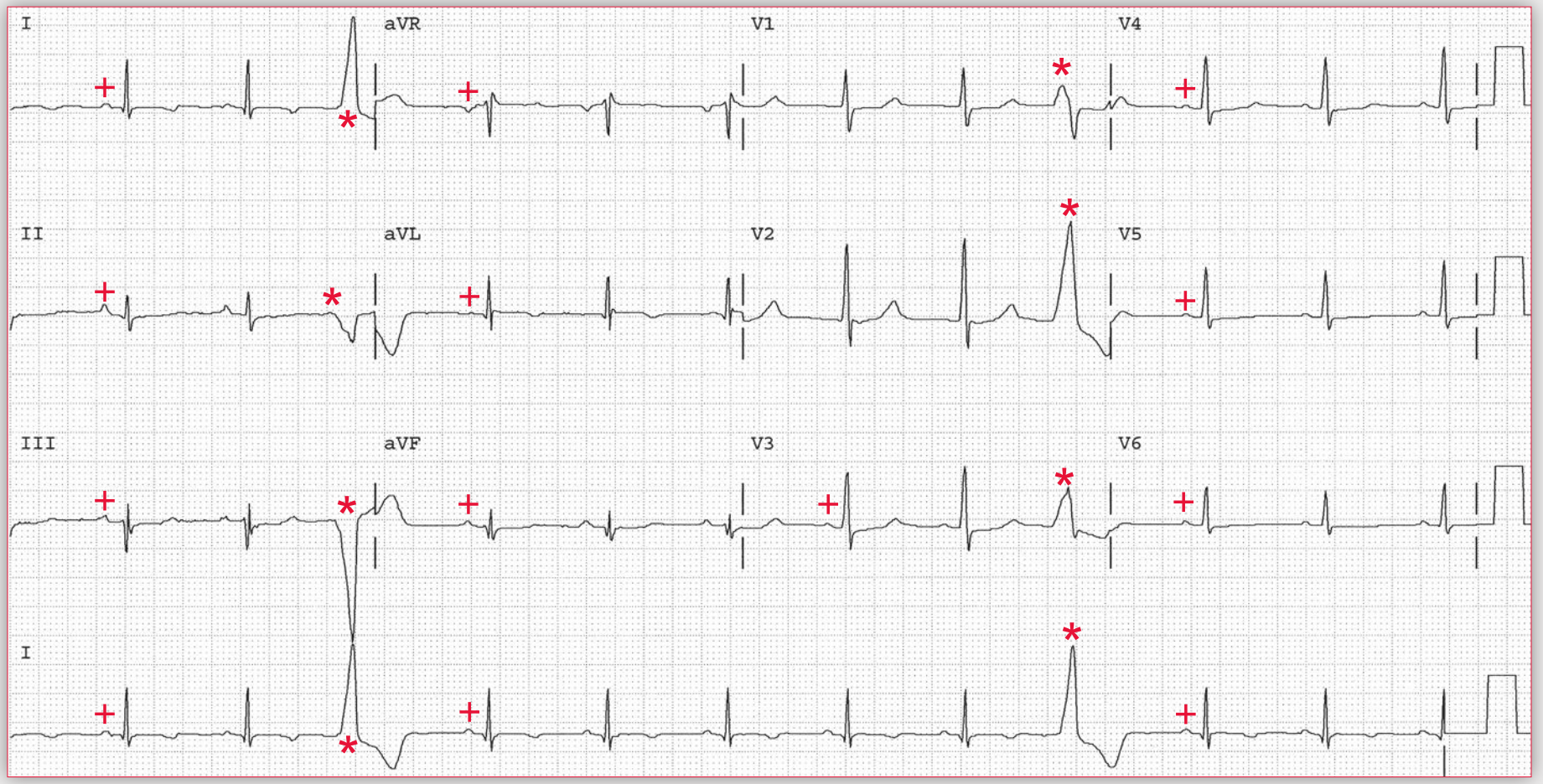


ECG 103B Analysis: Normal sinus rhythm, unifocal premature ventricular contractions, old inferoposterior wall myocardial infarction

The rhythm in **ECG 103B** is mostly regular at a rate of 74 bpm. The QRS complexes have the same duration, morphology, and axis as seen in **ECG 103A**, although the QRS complex amplitude in the limb leads and lateral precordial leads (V4-V6) is higher. The QT/QTc intervals are normal (400/440 msec). There are P waves (**+**) (positive in leads I, II, aVF, and V4-V6) before each QRS complex with a stable PR interval (0.20 sec). This is, therefore, a normal sinus rhythm. There are two premature (early) QRS complexes (*****) that are wide and without a preceding P wave. These are premature ventricular complexes.

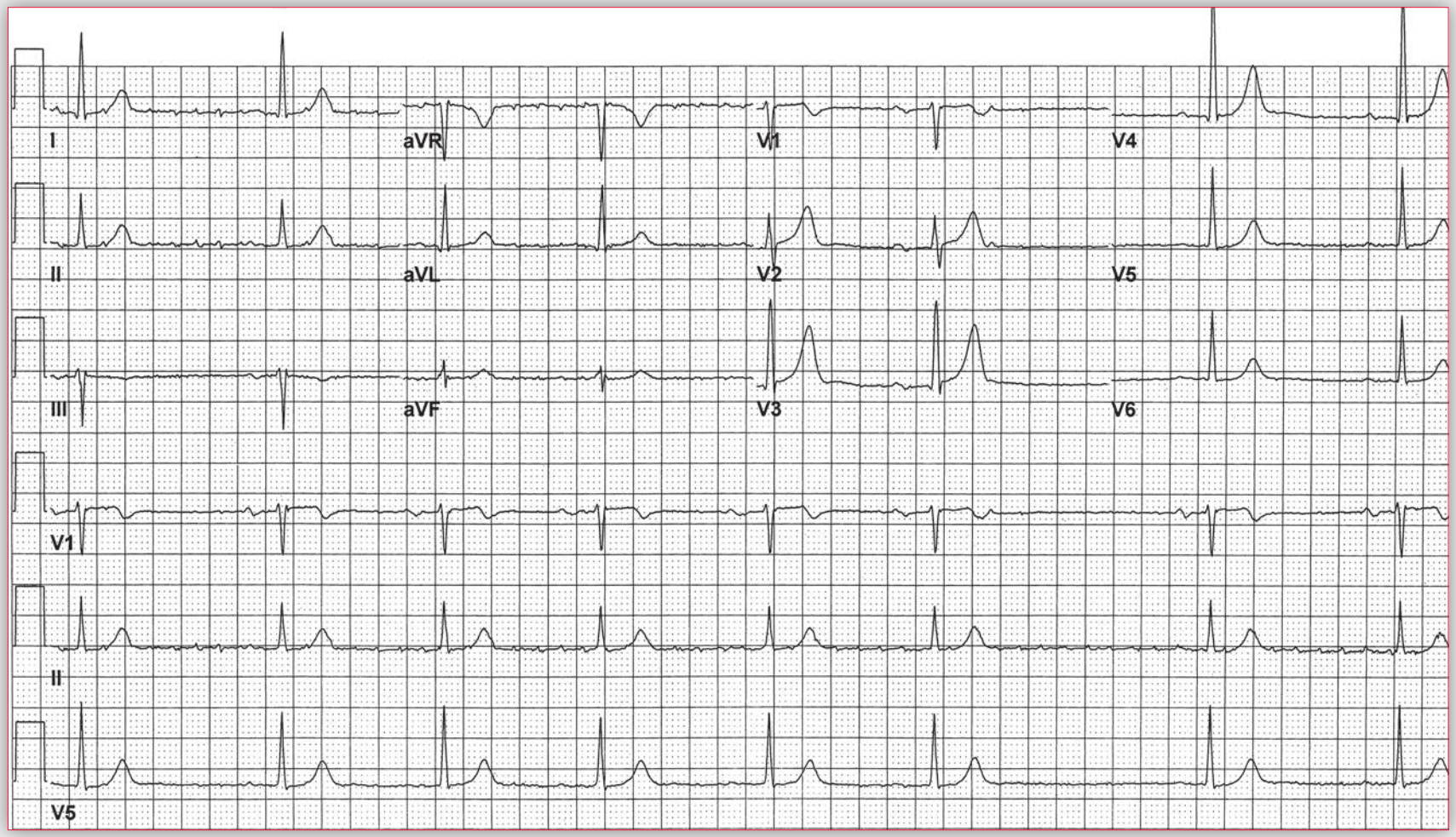
The ST-segment depressions and T-wave inversions in leads V1-V3 seen in **ECG 103A**, along with the clinical symptom of chest discomfort during tachycardia, are consistent with the presence of myocardial ischemia that is provoked by the elevated heart rate. These changes are not seen on **ECG 103B**, which is the baseline ECG for this patient. There is evidence of a prior inferoposterior infarction and hence the patient has underlying coronary artery disease with evidence of inducible ischemia. However, it should be noted that ST-T wave abnormalities may also be seen during tachycardias in the absence of coronary disease owing to derangements in cellular ion flux during the rapid depolarization–repolarization cycles. ■

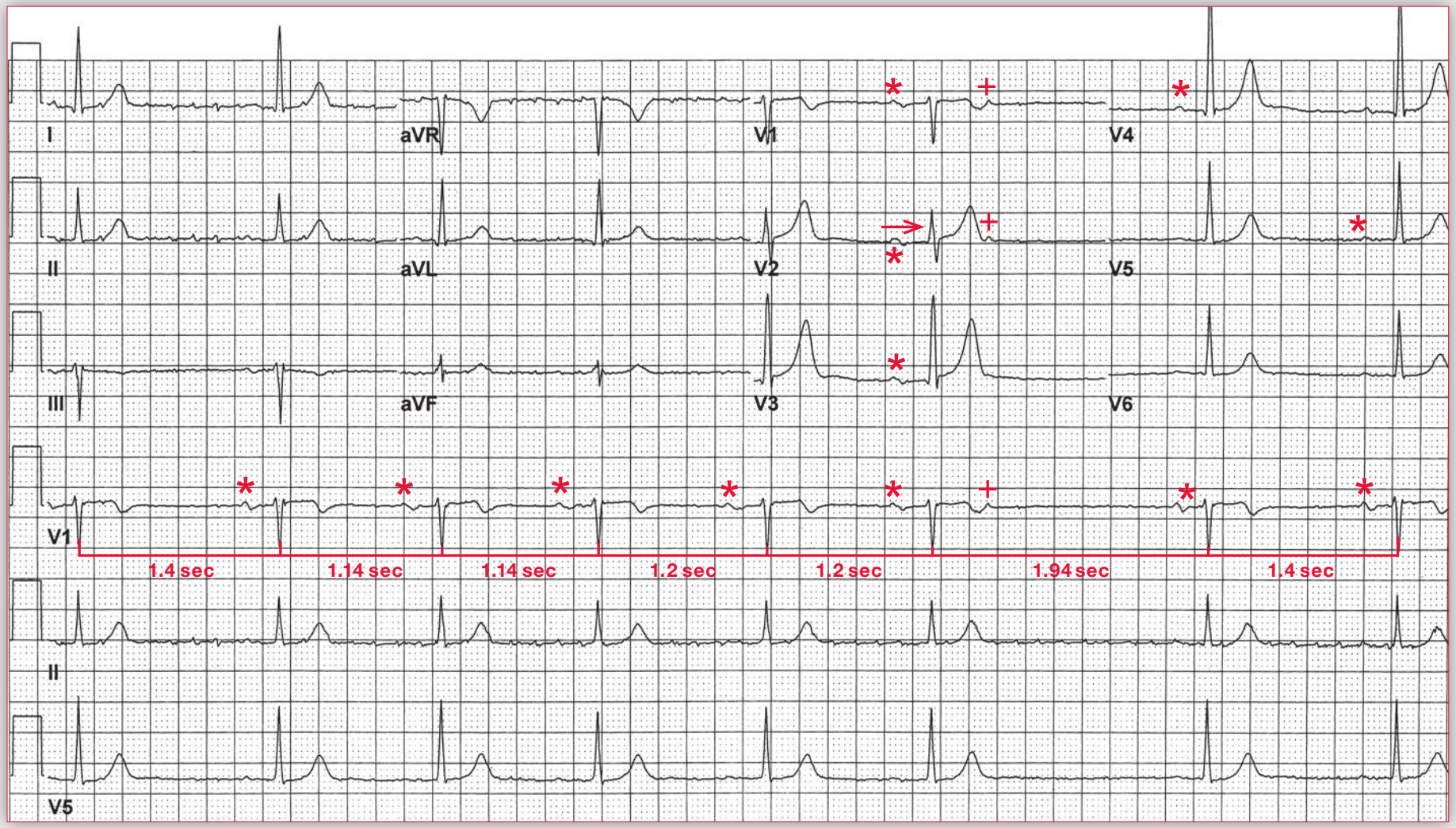
Notes

Practice Case 104

A 41-year-old man is being observed in the postoperative recovery room. He is noted to have moderate bradycardia and several dropped beats. He is asymptomatic.

What does his ECG show?





ECG 104 Analysis: Sinus bradycardia, sinus arrhythmia, first-degree AV block, blocked (nonconducted) premature atrial complexes

The rhythm is irregularly irregular at a rate of 48 bpm. There are three supraventricular rhythms that are irregularly irregular. These are (1) sinus arrhythmia in which there is one P-wave morphology and PR interval, (2) multifocal atrial rhythm (wandering atrial pacemaker) with a rate less than 100 bpm or multifocal atrial tachycardia with rate greater than 100 bpm in which there are three or more different P-wave morphologies and no P-wave morphology is dominant, or (3) atrial fibrillation, in which there is no organized atrial activity but there are fibrillatory waves. A P wave (*) precedes each QRS complex, and the P-wave morphology and PR interval are stable (0.26 sec). The P wave is positive in leads I, II, aVF, and V4-V6. This is, therefore, sinus bradycardia with first-degree AV block. The irregularity is the result of sinus arrhythmia. Although the rhythm is irregularly irregular with variability in the RR interval, the pause after the sixth QRS complex is substantially longer. Seen after the T wave of the sixth QRS complex is a superimposed P wave that is premature (+) and nonconducted. This represents a blocked or nonconducted premature atrial complex (PAC).

The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). The QT/QTc intervals are normal (400/360 msec). There is a tall R wave (→) in lead V2 that is the result of early transition or counterclockwise rotation in the horizontal plane. This is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, left ventricular forces develop earlier and are noted in the early precordial leads.

PACs are common and benign; they have no clinical importance. Because the P wave is nonconducted, however, there is a long RR interval. If nonconducted or blocked PACs were to become frequent, the effective heart rate would be much slower and might be associated with symptoms seen with bradycardia.

In this case the nonconducted P wave, which has a coupling interval of 0.72 second from the previous P wave (*ie*, a rate of 85 bpm), is nonconducted as a result of a relatively long refractory period and slow conduction through the AV node. These are related to the bradycardia (due to enhanced vagal tone) as well as intrinsic AV nodal disease (accounting for the prolonged PR interval). ■

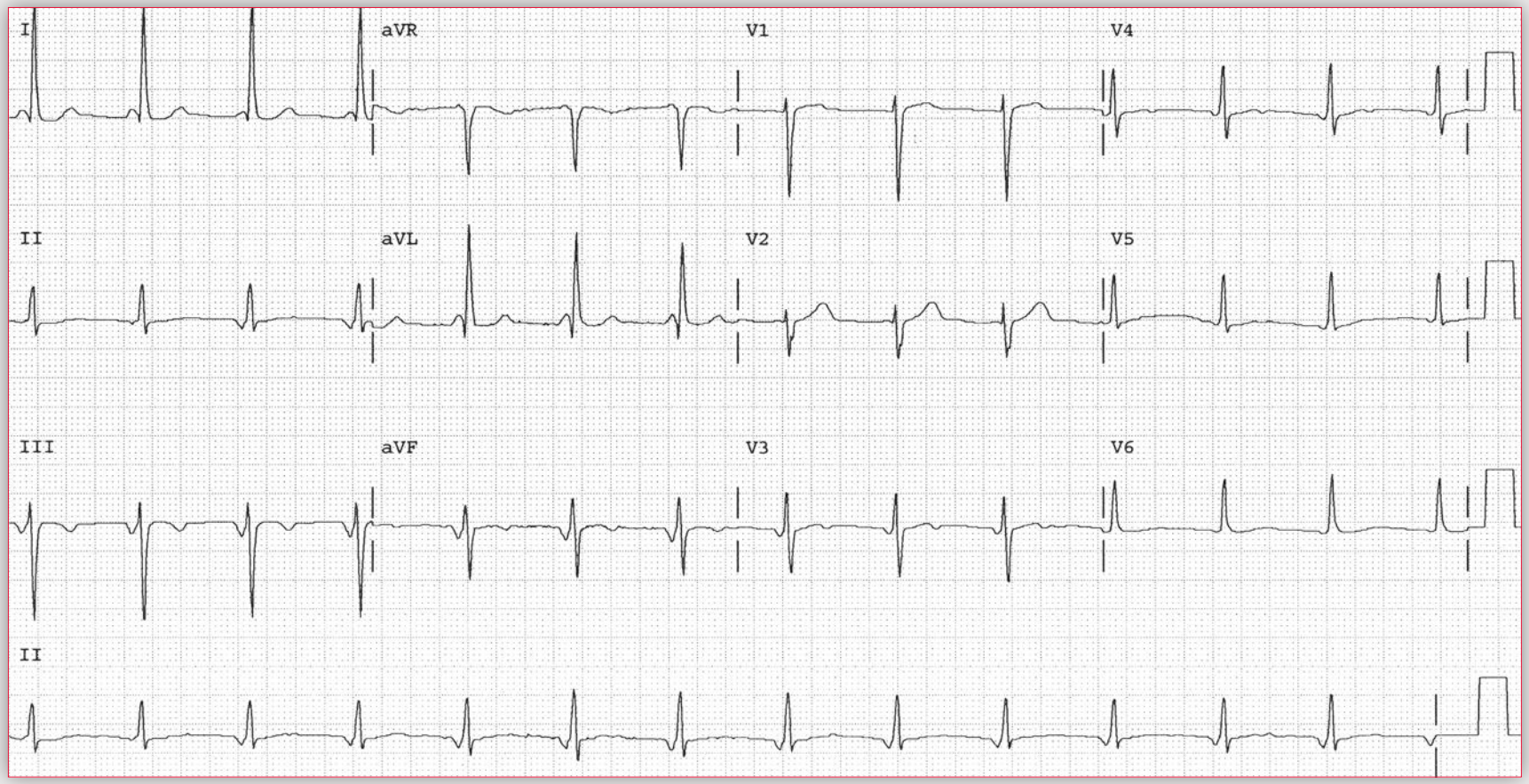
Notes

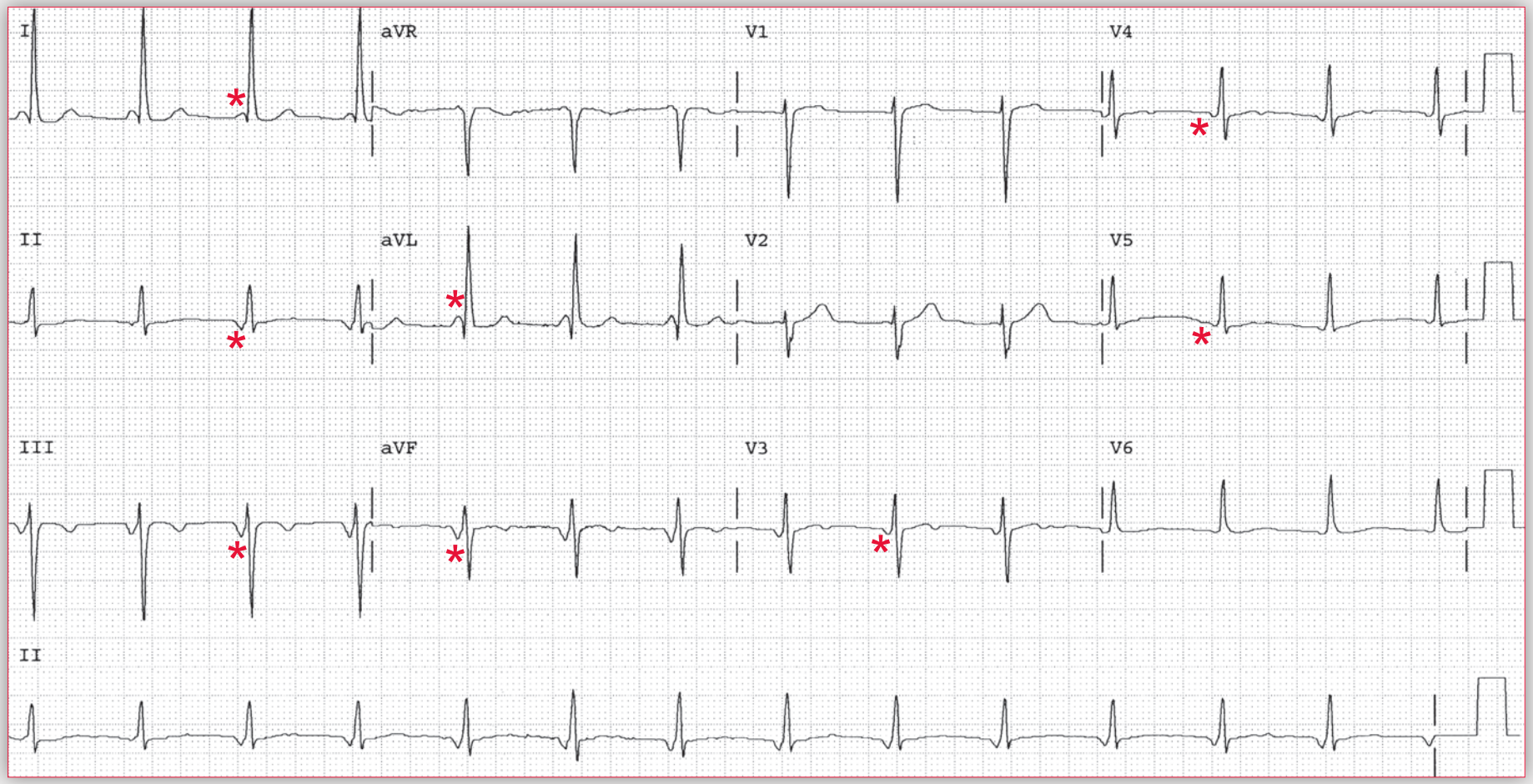
Practice Case 105

A 78-year-old man with sick sinus syndrome presents for routine follow-up. He has had no recent tachycardic or bradycardic episodes and feels generally well.

What does his ECG show?

What is the underlying mechanism?





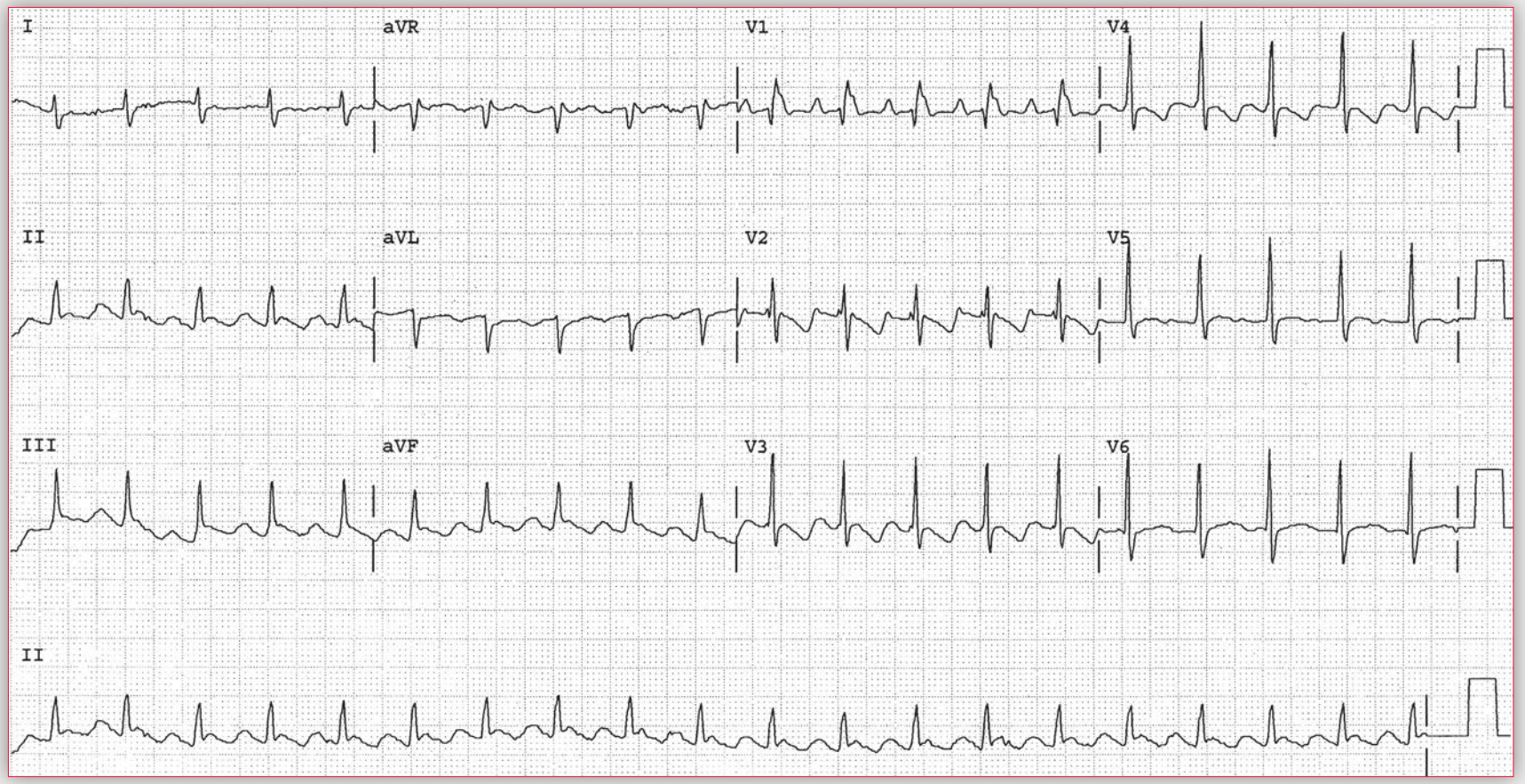
ECG 105 Analysis: Ectopic atrial rhythm, left axis, left ventricular hypertrophy

There is a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex with a stable but short PR interval (0.10 sec). The P wave is negative (inverted) in leads II, aVF, and V4-V6. This is, therefore, an ectopic atrial rhythm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis in the frontal plane is leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF); this is a physiologic left axis. The QT/QTc intervals are normal (360/420 msec). The QRS complex amplitude is increased in leads I (20 mm) and aVL (17 mm), consistent with left ventricular hypertrophy. Ectopic atrial rhythm, which results from an ectopic focus in the atrium, may be slightly more prevalent in patients with sinus node dysfunction. If the sinus node automaticity is decreased or fails to generate an impulse, an ectopic atrial pacemaker (which is usually suppressed or overdriven by sinus node activity) may become clinically evident. ■

Practice Case 106

A 14-year-old boy is brought to his pediatrician's office after he complains of being more tired than usual during a track meet. Many members of his family have hypertrophic cardiomyopathy, but the patient himself has no prior medical or cardiovascular

ECG 106A



Practice Case 106

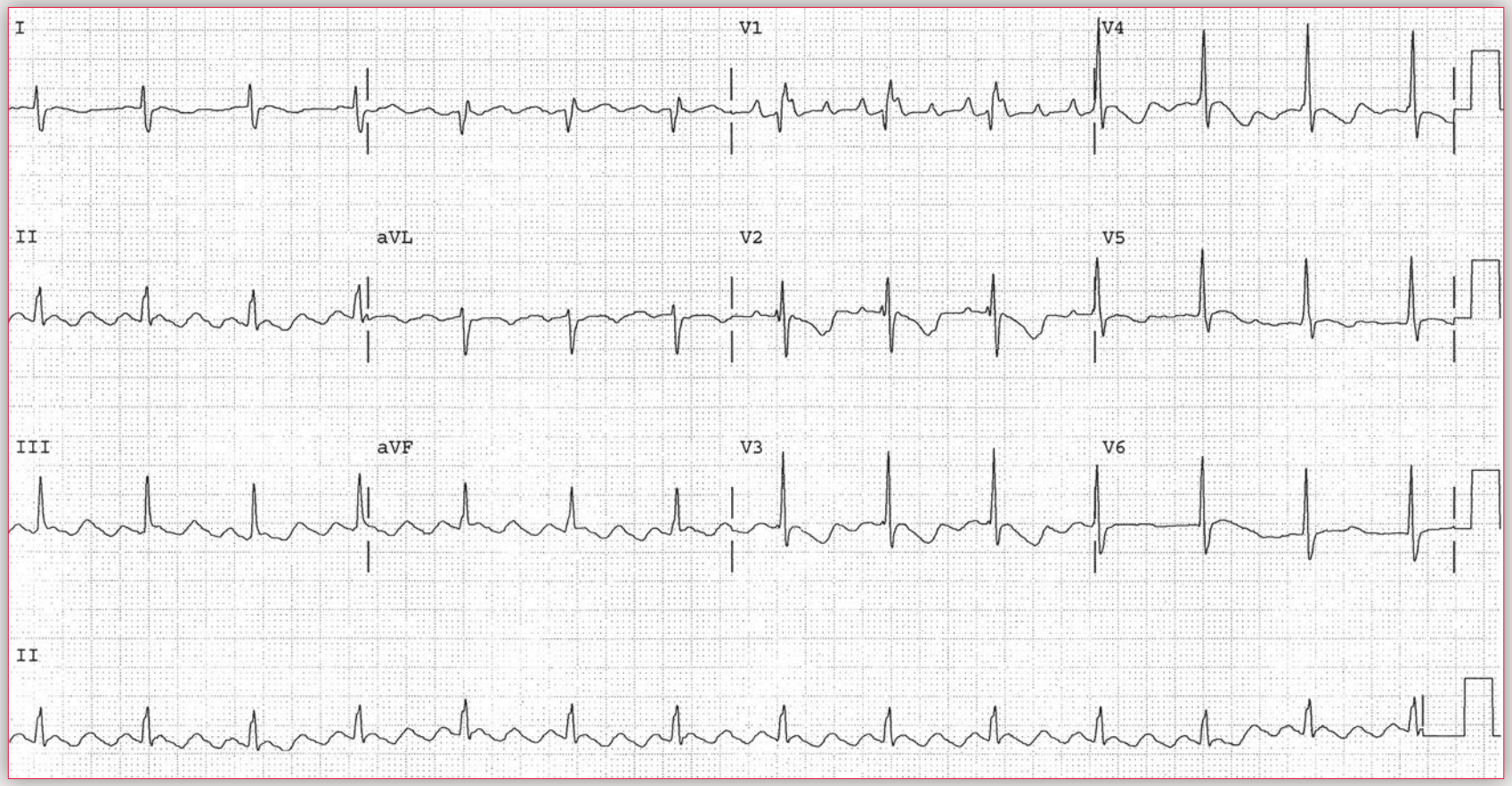
diagnosis. His heart rate on initial exam is 130 bpm; other vital signs and physical exam findings are normal. His initial ECG is shown (ECG 106A). Based on this ECG, he is sent to a local emergency room where a follow-up ECG is obtained (ECG 106B).

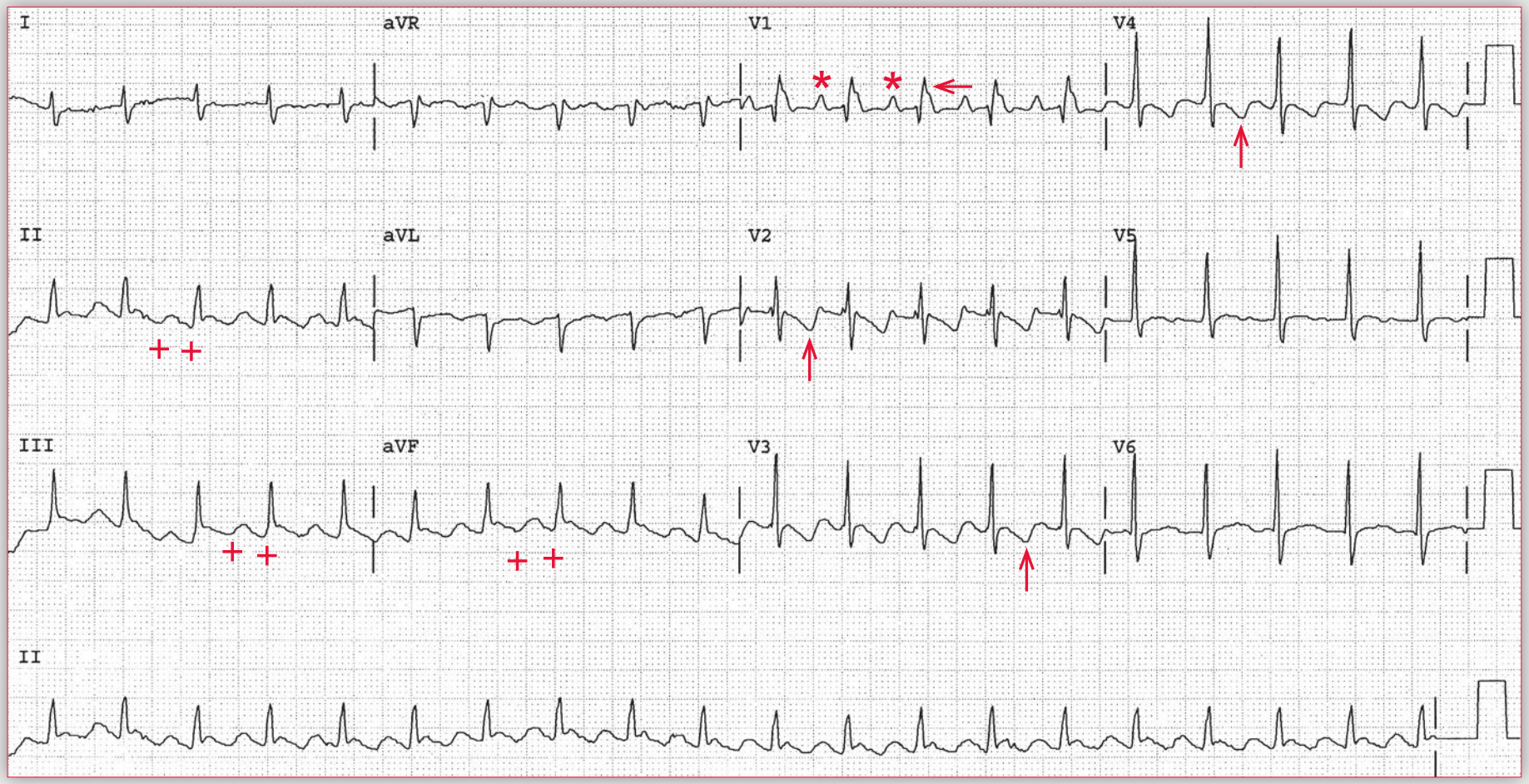
What is the etiology for the arrhythmia, and what features are useful for diagnosis?

Why is there variability in the ventricular rate?

What further workup is indicated?

ECG 106B





ECG 106A Analysis: Atrial flutter with 2:1 conduction, nonspecific ST-T wave abnormalities

ECG 106A shows a regular rhythm at a rate of 130 bpm. The QRS complex has a normal duration (0.10 sec), although in lead V1 it appears to be longer 0.14 sec), and has a morphology suggestive of right bundle branch block (RSR′ morphology in lead V1). However, the S waves in leads I and V5-V6 are not as wide as the R′ waveform in lead V1 (←). The axis is normal (+90°), with a biphasic QRS complex in lead I and a positive QRS complex in lead aVF. Although no distinct P waves are seen in leads II, III, and aVF, the baseline appears to have undulations (saw-tooth–like) that are suggestive of flutter waves (+). In lead V1 a distinct atrial waveform can be seen before each QRS complex (*). The terminal portion of the R′ waveform in lead V1 has obvious notching (↓), suggesting another atrial waveform. Indeed, when the intervals are measured they are all regular. The QT/QTc intervals are normal (280/410 msec). In addition, there are diffuse T-wave abnormalities and the T wave is inverted in leads V2-V4 (↑).

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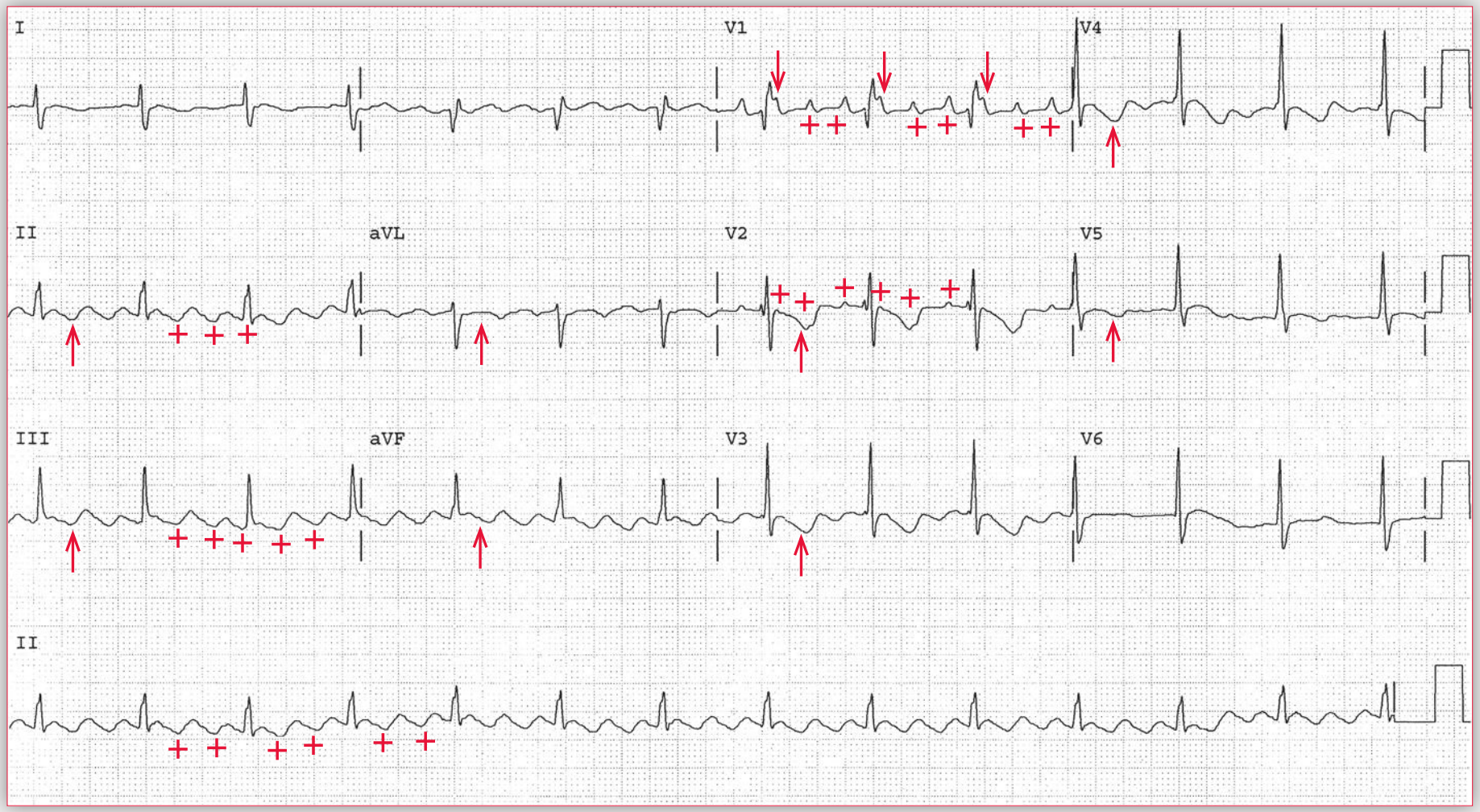


ECG 106B Analysis: Atrial flutter with 3:1 conduction, nonspecific ST-T wave abnormalities

The QRS complex duration, morphology, and axis in **ECG 106B** are the same as in **ECG 106A**. The rhythm is regular, and the rate is now 84 bpm. Obvious atrial flutter waves (+) with a saw-tooth pattern are now apparent in leads II, III, aVF, and V1. In lead V1, two distinct atrial waveforms can be seen between each QRS complex. By measuring the intervals it can be seen that an on-time third atrial waveform is on the downstroke of the R′ waveform (↓), as was assumed to be the case in **ECG 106A**. It is noted that the atrial rate is 260 bpm and there is continuous undulation of the baseline between each atrial waveform. The only atrial arrhythmia with a regular atrial rate ≥260 bpm is atrial flutter. In addition, the continuous electrical activity with undulations between each atrial waveform and no isoelectric baseline is characteristic of atrial flutter. On this ECG there is 3:1 AV block or AV conduction. **ECG 106A** shows the same atrial rate of 260 bpm and there is atrial flutter with 2:1 AV block. Diffuse T-wave inversions are present (↑), as noted on **ECG 106A**.

It is uncommon for atrial flutter to be associated with AV block with an odd ratio, especially when there is also an AV block with an even ratio. This suggests that there are two levels of block within the AV node.

Atrial flutter is an uncommon rhythm in young individuals and may be the manifestation of an underlying cardiomyopathy. This is of particular concern given the family history of hypertrophic cardiomyopathy (HCM). It is important to obtain an echocardiogram for further evaluation. If HCM is diagnosed, more aggressive therapy would be indicated for the atrial flutter because flutter is often associated with significant symptoms in these patients. It may also precipitate atrial fibrillation. These atrial arrhythmias are associated with significant hemodynamic symptoms in patients with HCM.

Importantly, if this patient has HCM his risk for sudden death must be assessed, particularly because he is young and athletic. In the United States, HCM is one of the leading cause of sudden cardiac death in young athletes. Factors associated with an increased risk for sudden cardiac death include the following:

- A previous episode of sudden death, sustained ventricular tachycardia, or syncope
- A family history of sudden cardiac death
- Significant left ventricular hypertrophy (> 3 cm width of myocardium)
- Exercise-induced hypotension
- Presence of nonsustained ventricular tachycardia on monitoring

The presence of HCM on an echocardiogram would necessitate an exercise test and ambulatory monitoring for further assessment. ■

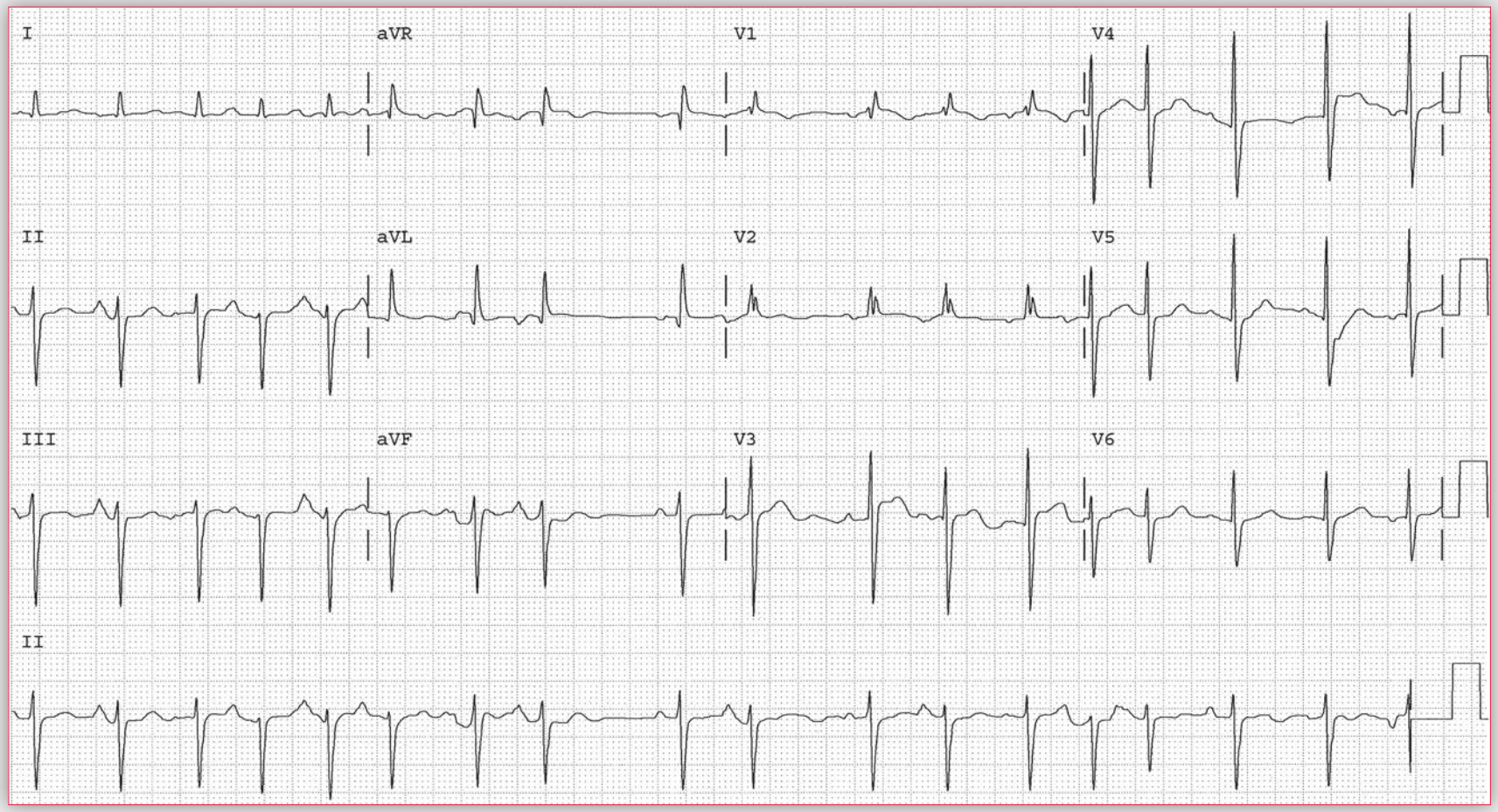
Notes

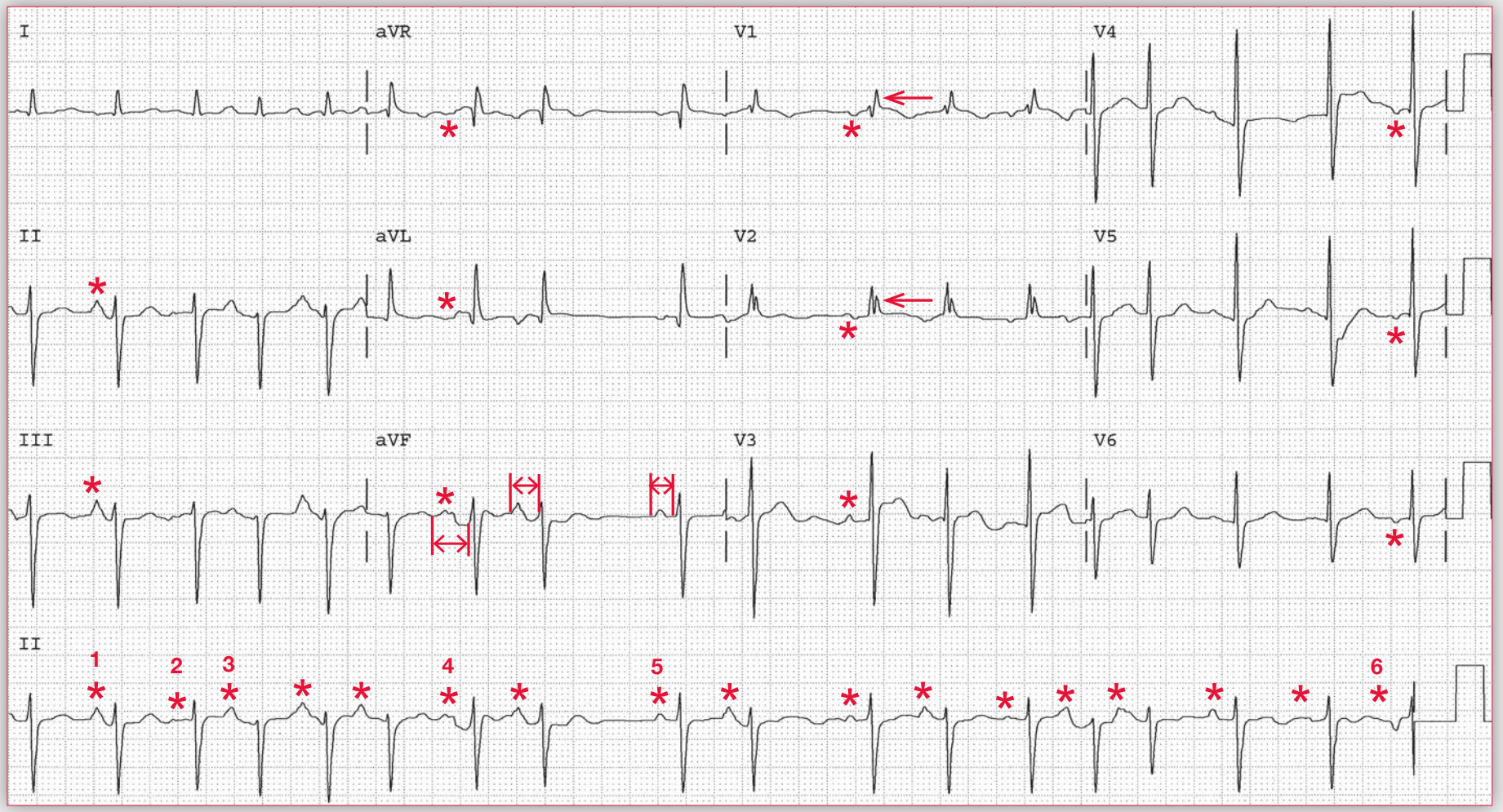
Practice Case 107

A 36-year-old woman with primary pulmonary hypertension is seen in her physician's office after initiation of pulmonary vasodilator therapy. She is found to be tachycardic with an irregularly irregular heart rate.

What does her ECG show?

What therapy is indicated?





ECG 107 Analysis: Multifocal atrial tachycardia, left anterior fascicular block

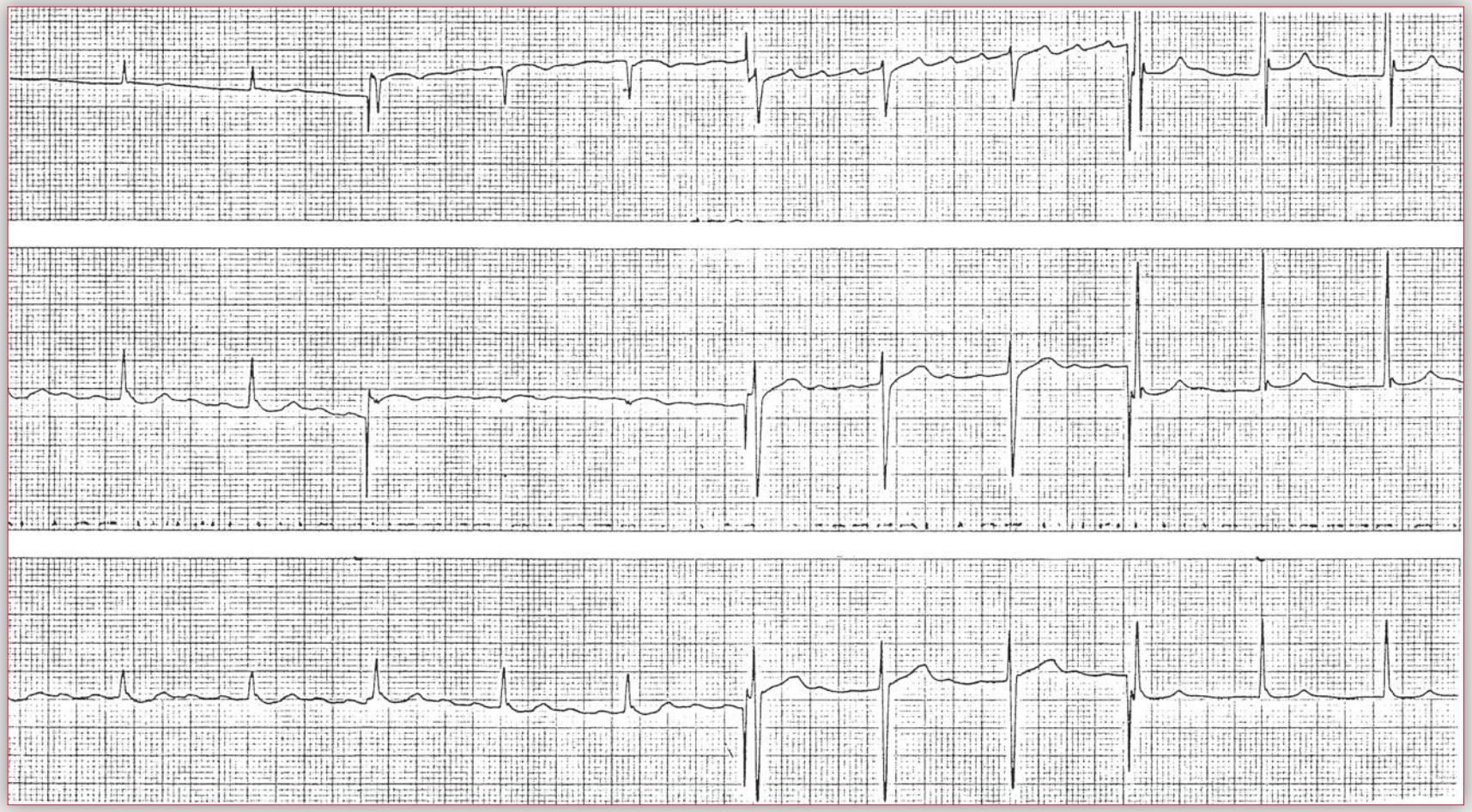
The rhythm is irregularly irregular at an average rate of 108 bpm. There are only three supraventricular arrhythmias that are irregularly irregular: (1) sinus arrhythmia in which there is one P-wave morphology and PR interval, (2) multifocal atrial rhythm (wandering atrial pacemaker) with a rate less than 100 bpm or multifocal atrial tachycardia with a rate greater than 100 bpm in which there are three or more different P-wave morphologies and no P-wave morphology is dominant, or (3) atrial fibrillation, in which there is no organized atrial activity but there are fibrillatory waves. There is atrial activity, and a P wave (*) is seen before each QRS complex. However, at least three different P-wave morphologies (and no one dominant morphology) can be seen as well as variability in the PR interval (↔). Hence this is multifocal atrial tachycardia. The QRS complexes are of normal duration (0.08 sec). The axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). The two causes for an extremely leftward axis are an old inferior wall myocardial infarction with deep Q waves in leads II and aVF or a left anterior fascicular block with an rS morphology in leads II and aVF; this is left anterior fascicular block. There is an R′ waveform in leads V1-V2 (←) associated with a normal QRS complex duration; this indicates a right ventricular conduction delay, which is a normal variant. The QT/QTc intervals are prolonged (360/480 msec), possibly as a result of drug therapy.

Multifocal atrial tachycardia is often seen in association with pulmonary disease, including pulmonary hypertension, as well as heart failure. Multifocal atrial tachycardia may occasionally revert to atrial fibrillation. Initial therapy is directed at controlling symptoms, which are usually the result of a rapid ventricular response rate. Hence AV nodal blocking agents are useful for symptom relief. Treatment of the arrhythmia itself is more challenging. The first approach is to treat the associated underlying medical condition. There is some evidence of benefit for potassium or magnesium replacement if either hypokalemia or hypomagnesemia is present. Data regarding the efficacy of verapamil or β-blockers are limited. The standard anti-arrhythmic agents are not of proven benefit. ■

Practice Case 108

A patient is admitted to the hospital for initiation of therapy with a class IC anti-arrhythmic drug. His admission ECG is shown (ECG 108A). Following drug loading, a second ECG (108B) is obtained.

ECG 108A

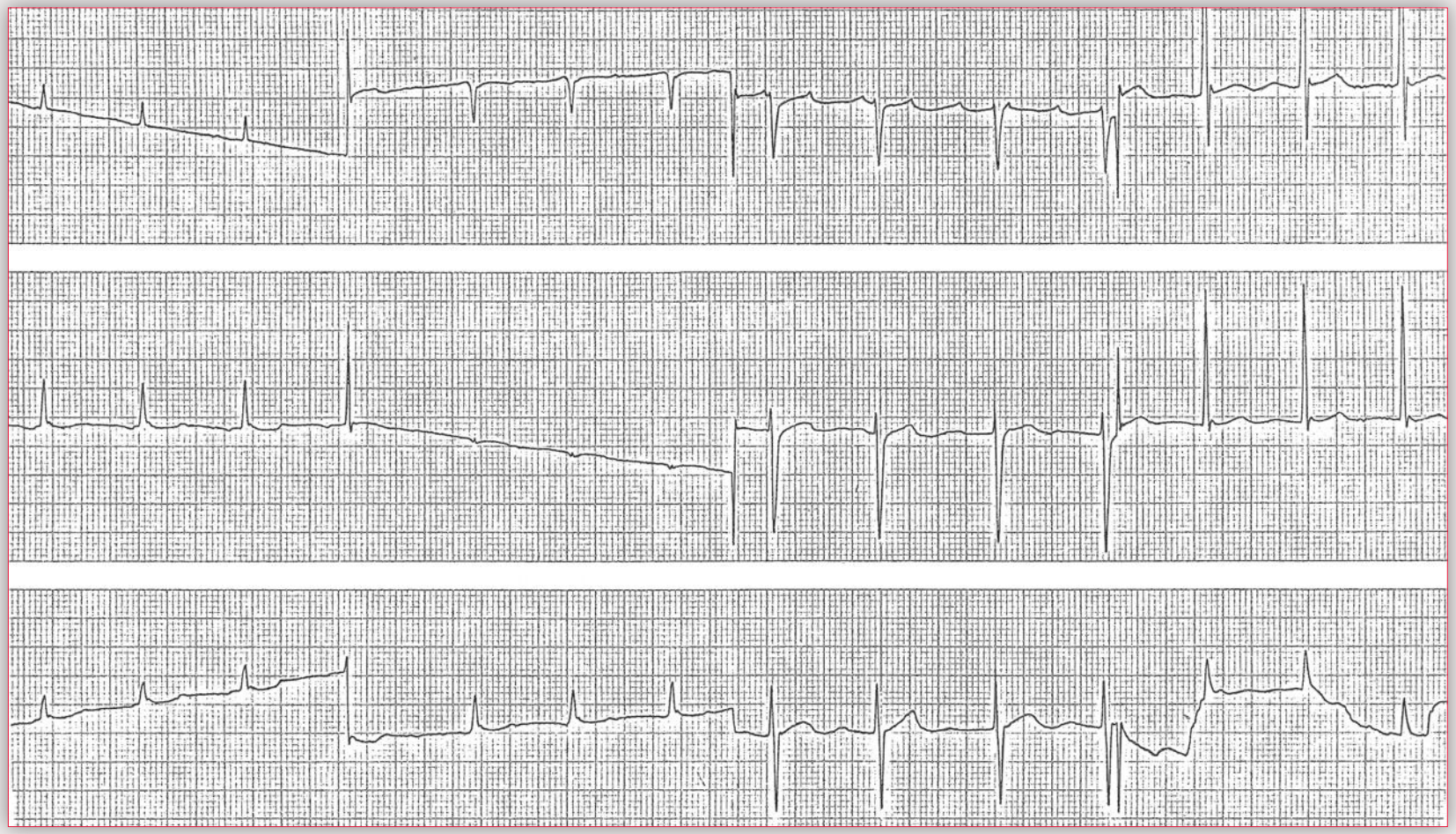


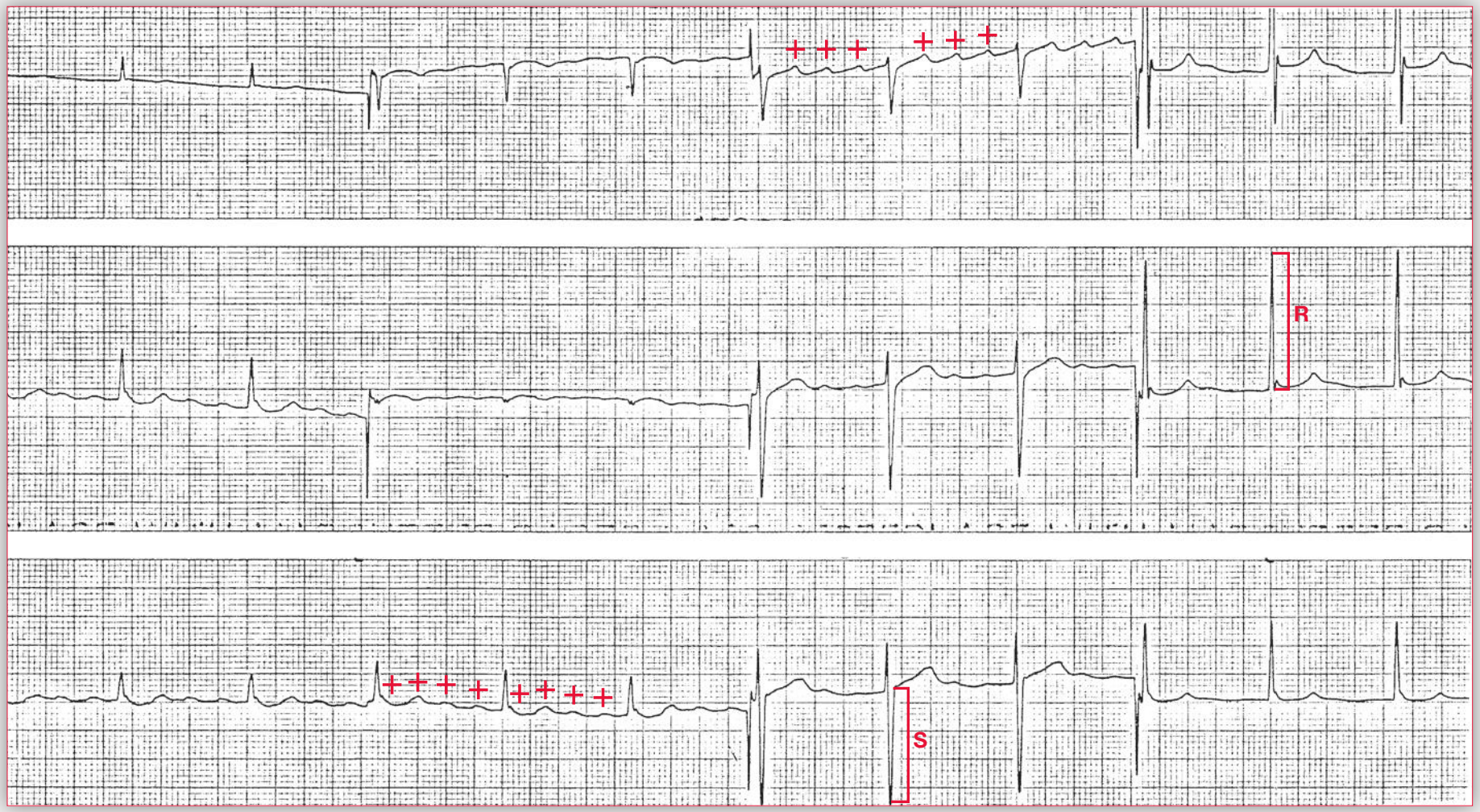
Practice Case 108

For what is the patient is admitted?

What is the effect of a class IC agent on this arrhythmia?

ECG 108B





ECG 108A Analysis: Atrial flutter with 4:1 AV block or conduction, left ventricular hypertrophy

ECG 108A shows a regular rhythm at a rate of 70 bpm. The QRS complexes have a normal duration (0.08 sec) and morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/430 msec). Noted are atrial flutter waves, which are most prominent in lead V1 (+) but are also seen in leads II, III, and aVF. The waveforms are continuously undulating, with a saw-tooth pattern, at a rate of 280 bpm. This is, therefore, atrial flutter with 4:1 AV conduction. The QRS complex amplitude is increased in leads II and aVF; the S-wave depth in lead V3 is 20 mm and the R-wave amplitude in lead V5 is 25 mm (S-wave depth in lead V3 + R-wave amplitude in lead V5 = 45 mm). This is consistent with a diagnosis of left ventricular hypertrophy (*ie*, S-wave depth in lead V3 + R-wave amplitude in lead V5 ≥ 35 mm).

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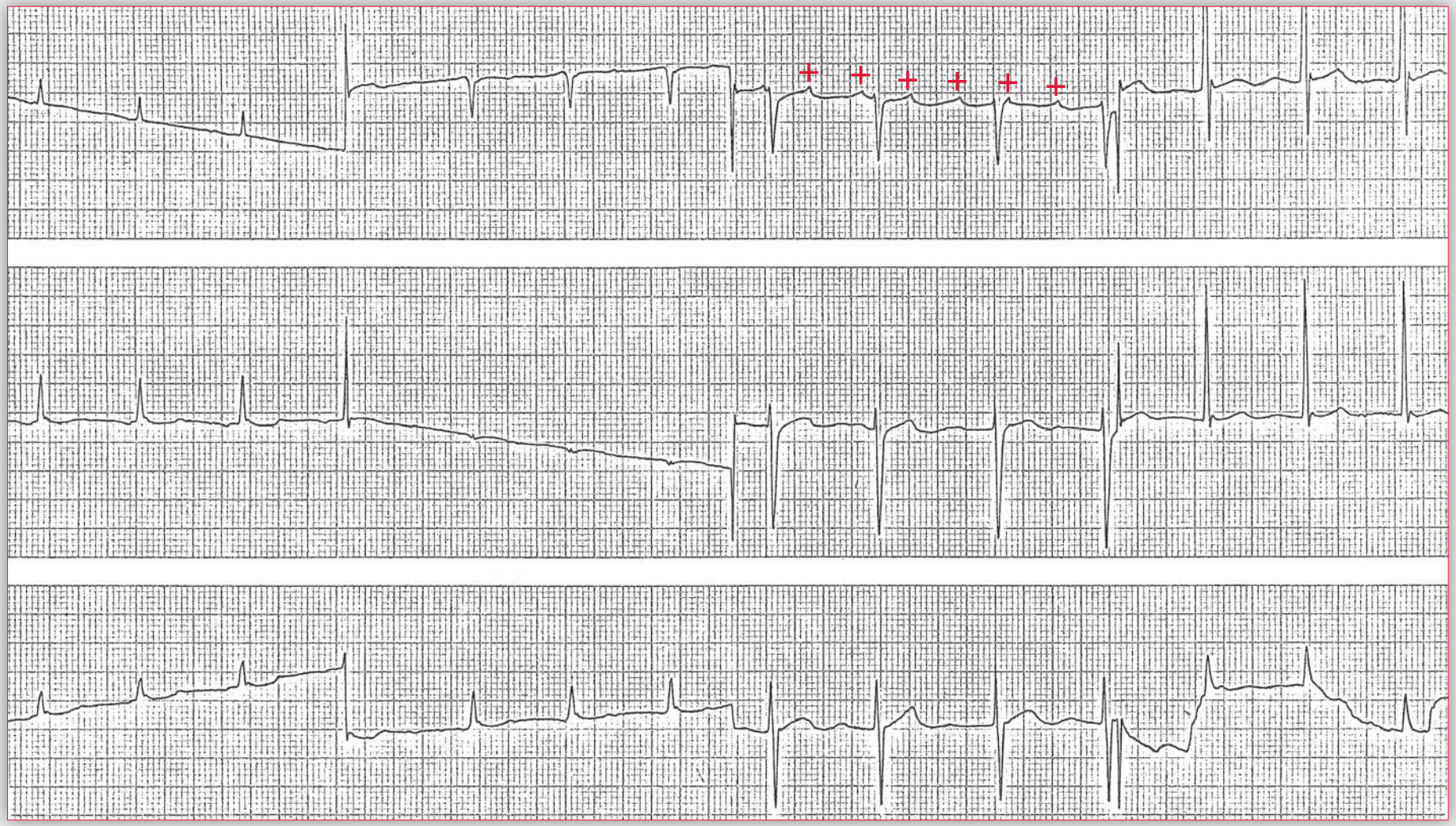


ECG 108B Analysis: Slow atrial flutter (with 2:1 AV block or conduction) due to class IC antiarrhythmic drug therapy (sodium channel blockade)

ECG 108B was obtained after several days of therapy with propafenone, a class IC agent. The rhythm is regular at a rate of 75 bpm. The QRS complexes have the same duration, axis, and morphology as those in **ECG 108A**. The QT/QTc intervals are the same as in **ECG 108A**. Regular undulations of the atrial waveforms can be seen, especially in lead V1 (+), at an atrial rate of 150 bpm. The waveforms have an undulating or saw-tooth pattern. Although the atrial flutter rate is slower as a result of the anti-arrhythmic drug therapy, the drug does not alter the morphology of the flutter waveforms. Hence this is atrial flutter with 2:1 AV conduction.

The atrial flutter rate is between 260 and 320 bpm. However, disease of the atrial myocardium with areas of fibrosis can result in slowing of the atrial flutter rate as a result of changes that lead to slowing of impulse velocity. The use of a class IA, IC, or III anti-arrhythmic drugs can also result in a slowing of the atrial flutter rate. The class I agents significantly reduce the rate of upstroke of phase 0 of the action potential. This results in a slowing of impulse conduction velocity and hence slowing of the atrial flutter rate but no change in the morphology of the flutter waveforms. The class III antiarrhythmic drugs prolong the myocardial refractory period. This will also result in a slowing of the atrial flutter rate as the increase in refractoriness will also reduce the rate of impulse circulating around the reentrant circuit. ■

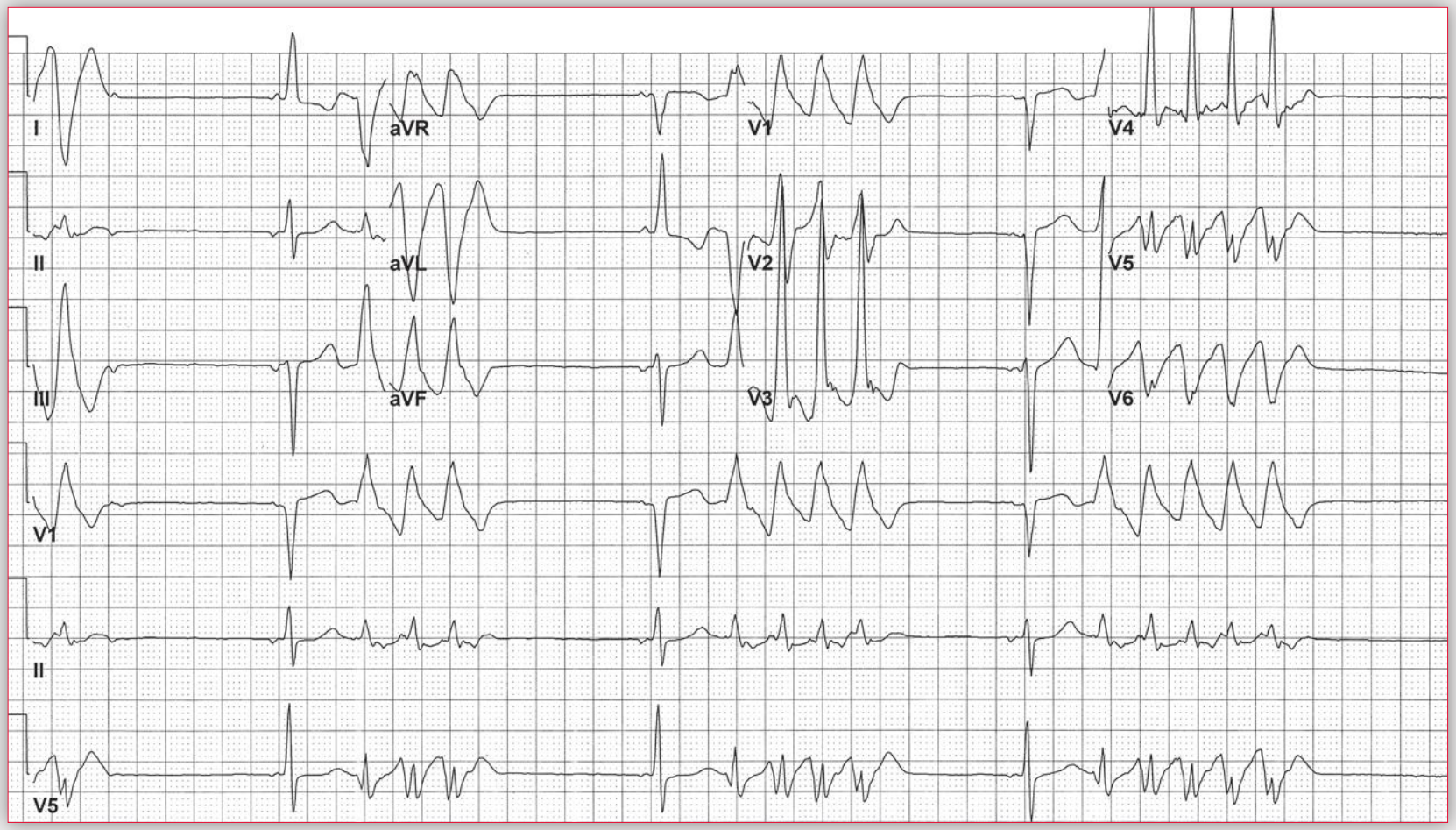
Notes

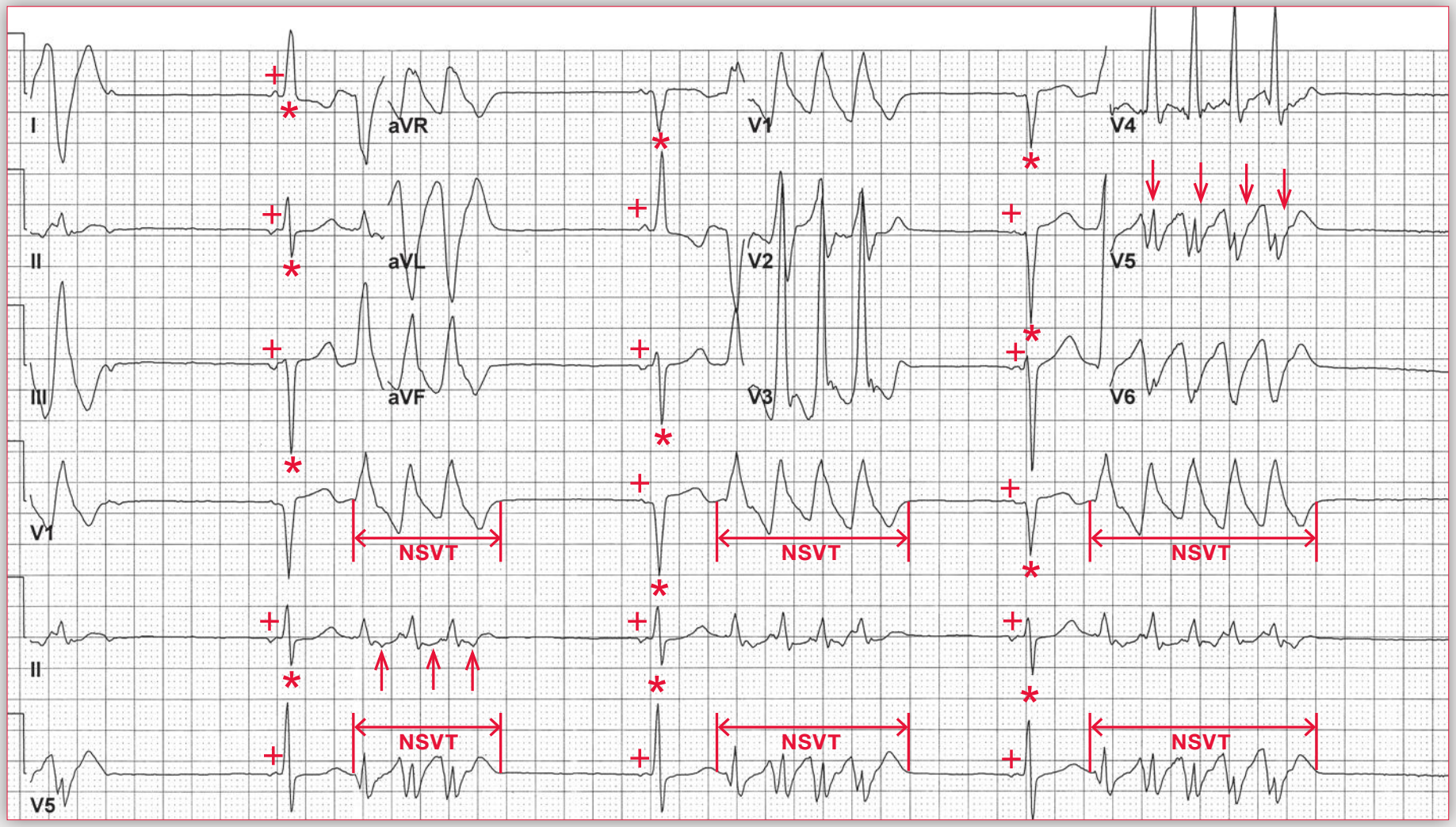
Practice Case 109

A 72-year-old man with past myocardial infarction, a left ventricular ejection fraction of 30%, and prior episodes of heart failure presents with intermittent palpitations. The following ECG is obtained while the patient is having symptoms.

What abnormality is causing his palpitations?

What is the anatomic origin of this ECG abnormality?





ECG 109 Analysis: Ectopic atrial rhythm, monomorphic nonsustained ventricular tachycardia

There are three narrow QRS complexes (0.10 sec) (*) that are preceded by a P wave (+) with a constant PR interval (0.14 sec). However, the P waves are negative in leads II and aVF and negative–positive in lead V5. They are, therefore, not sinus in origin but rather ectopic atrial beats. They have a left axis, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF).

After each narrow QRS complex there are episodes of three to five QRS complexes (↔), at a rate of 200 bpm. The QRS complexes are wide (0.16 sec) with an abnormal morphology. Although they have the same basic morphology, subtle differences can be seen in the QRS complexes (↓) and ST-T waves (↑). Therefore, the wide QRS complexes are ventricular and this is monomorphic nonsustained ventricular tachycardia, defined as more than three sequential QRS complexes lasting up to 30 seconds. Wide complex tachycardias are due to either ventricular tachycardia or supraventricular tachycardia with aberrancy (*ie*, rate-related or underlying bundle branch block). Other features in addition to the variability of QRS complex and ST-T wave morphology that point toward a ventricular origin for this wide complex tachycardia include a QRS complex width of 160 msec or greater and a QRS complex morphology that is not consistent with either a left (LBBB) or right (RBBB) bundle branch block, particularly the lack of an RSR′ pattern in lead V1 and an R-wave amplitude that is less than the S-wave depth in lead V6. Alternatively, an RSR′ pattern in lead V1 and an R-wave amplitude that is taller than the S-wave depth in lead V6 are the typical findings of supraventricular tachycardia with RBBB. In addition, there is an R/S complex in lead V2 and the R wave (> 100 msec) is wider than the S wave and it is greater than 100 msec in duration. This is consistent with a ventricular origin. The R wave that is wider than the S wave indicates that initial ventricular activation is abnormal, *ie*, a ventricular complex. With a rate-related aberration the initial forces of the QRS complex are normal (*ie*, the R-wave width is less than 100 msec and narrower than the S-wave amplitude), whereas with a ventricular complex the initial forces are slow as a result of activation that is not via the normal Purkinje fibers but rather directly through the ventricular myocardium.

The anatomic origin of ventricular tachycardia can be determined from a 12-lead ECG. If the R-wave amplitude is taller than the S-wave depth in lead V1 (R>S), then the tachycardia has an RBBB-like morphology and, therefore, most likely originates in the left ventricle. Leads in which the QRS complex is primarily negative in deflection represent the origin of the tachycardia (*ie*, the electrical vector is moving away from the origin and hence these leads). In this case, the QRS complex is negative in leads I and aVL; therefore, the ventricular tachycardia originates from the lateral wall of the left ventricle. The tachycardia is positive in leads II, III, and aVF and hence is inferiorly directed. ■

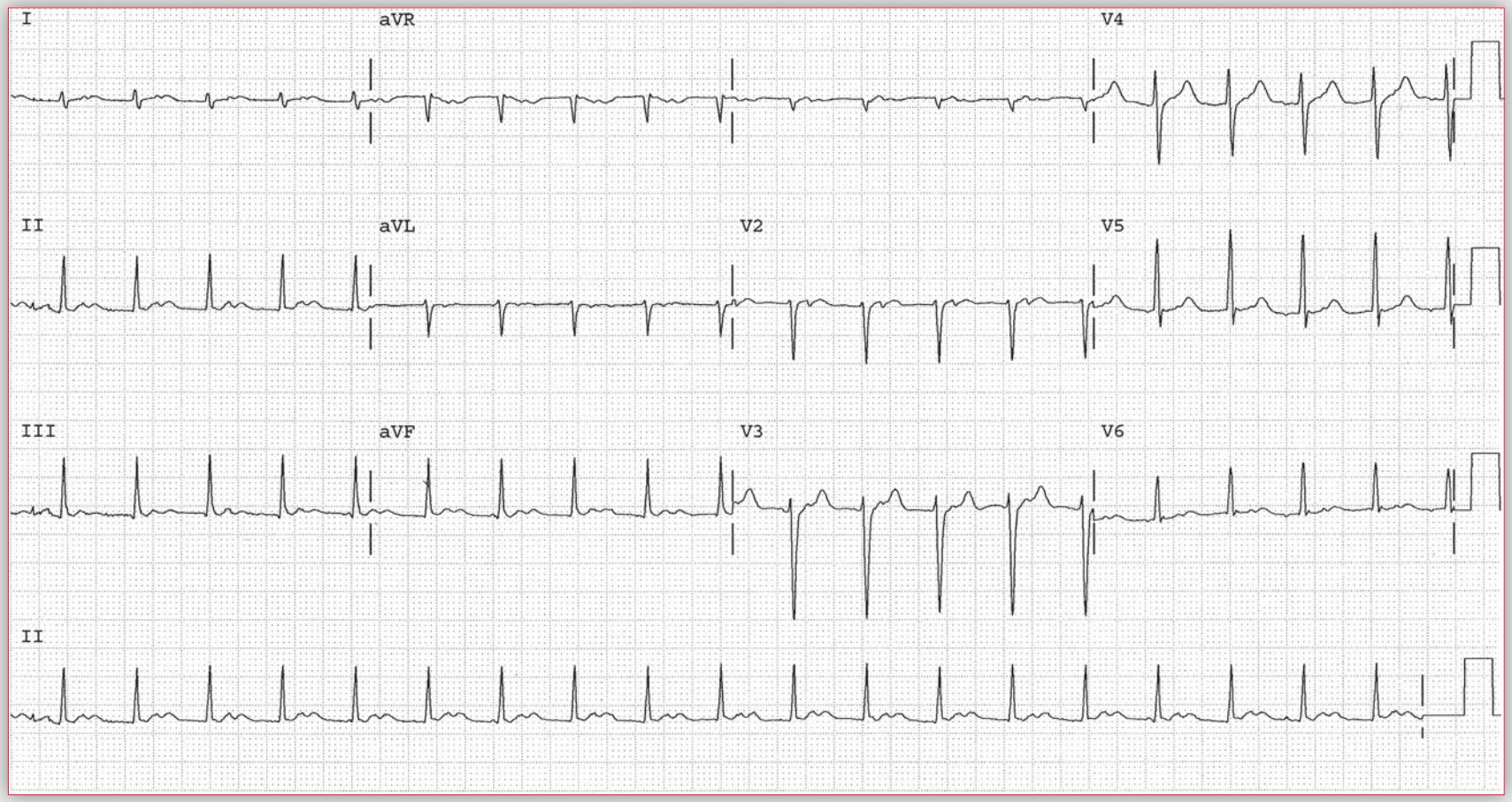
Notes

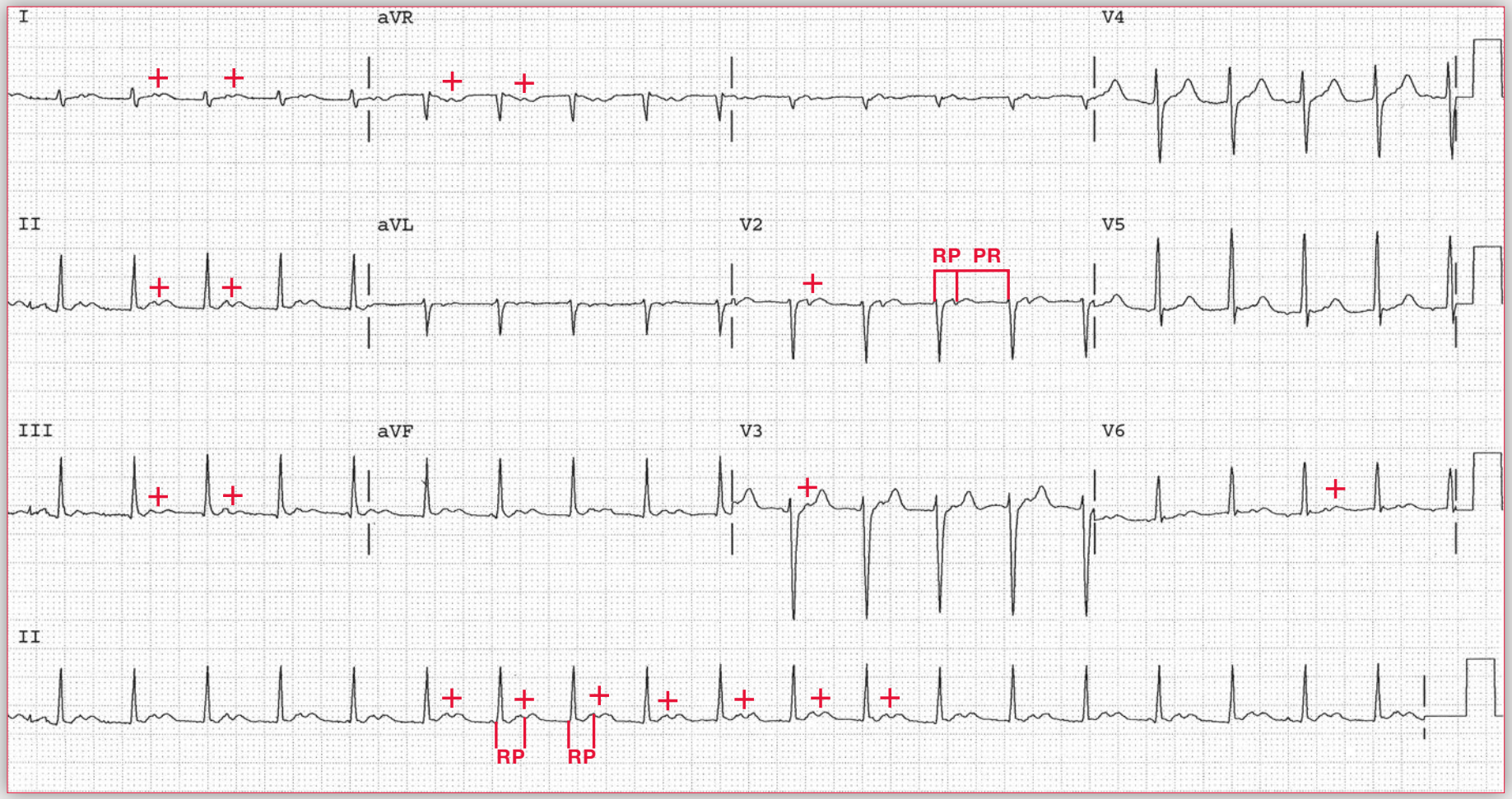
Practice Case 110

A 19-year-old woman presents to her primary care physician with complaints of palpitations and a racing heart. Symptoms often occur within an hour of her morning cup of coffee and resolve about 30 minutes later. She deliberately drank a large cup of coffee just prior to her appointment and is having symptoms during the appointment. An ECG is promptly obtained.

What abnormalities are notable on the ECG?

What therapy may be offered?





ECG 110 Analysis: Ectopic junctional tachycardia

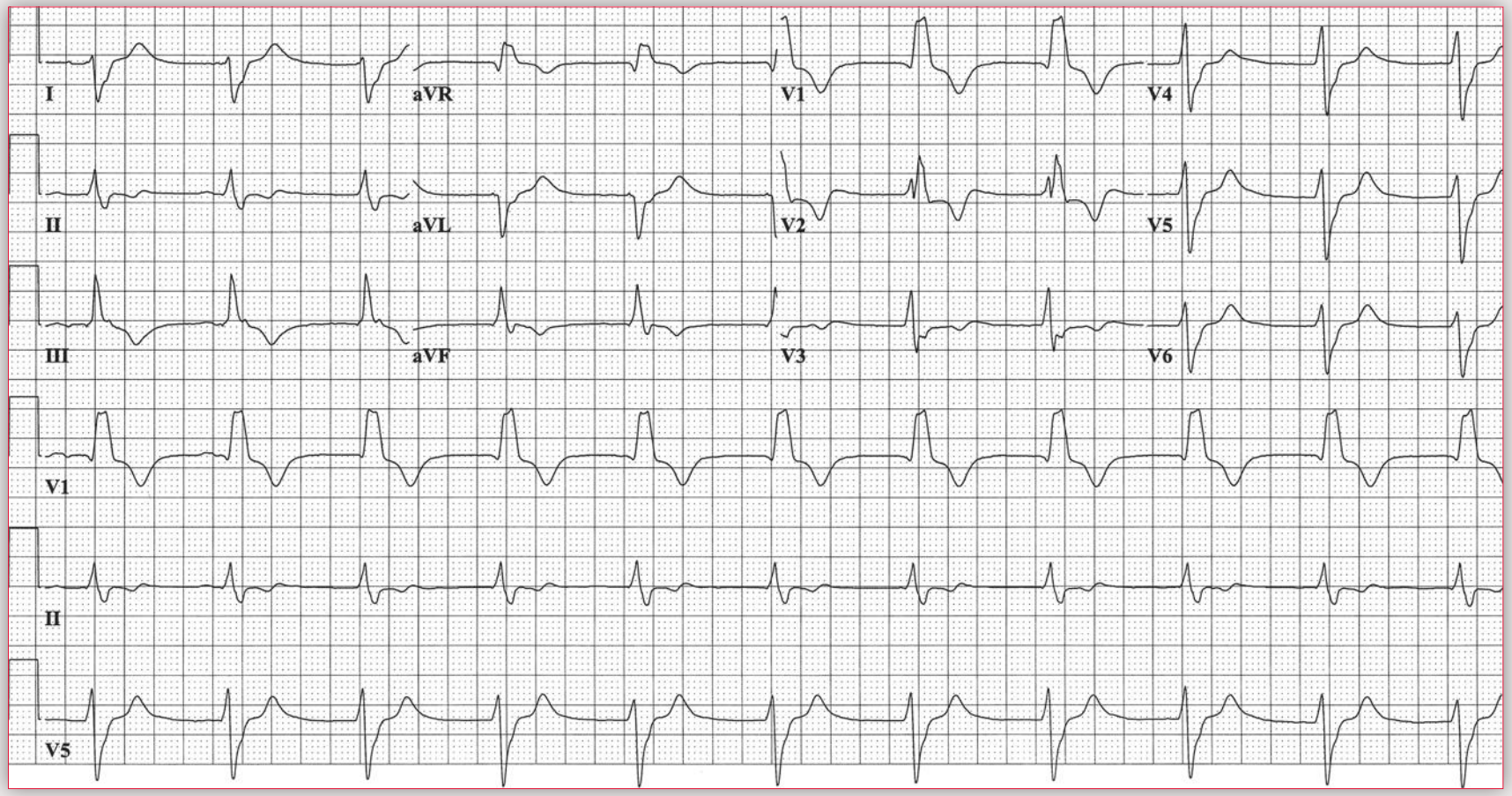
The rhythm is regular at a rate of 118 bpm. The QRS complexes have a normal duration (0.08 sec) and morphology; the axis is also normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/450 msec). There are no P waves before any of the QRS complexes. There is notching of the initial portion of the T waves (+), especially obvious in leads II, III, aVF, and V2-V3. The P waves are negative in leads II and aVF. These are retrograde P waves with a fixed RP interval (⊔). Hence this is ectopic junctional tachycardia.

If a clear trigger to a bothersome arrhythmia can be identified, avoidance of the trigger may be all that is required. In this case, the likely trigger is coffee, which contains caffeine, a known cardiac stimulant that has sympathomimetic activity. If more aggressive and potentially curative therapy is requested, an invasive electrophysiologic study may identify a discrete focus, region, or track that can be selectively modified or destroyed by ablation. Alternatively, the ectopic junctional focus can be suppressed by an AV nodal active agent such as a calcium channel blocker, beta blocker or digoxin. A standard class I or III antiarrhythmic agent may also be effective. ■

Practice Case 111

A 74-year-old man with diabetes, hypertension, and coronary artery disease complicated by a prior myocardial infarction (MI) is admitted with heart failure and found to have a left ventricular ejection fraction of 28%. An ECG is obtained (ECG 111A). ECG 111B is obtained the following day.

ECG 111A

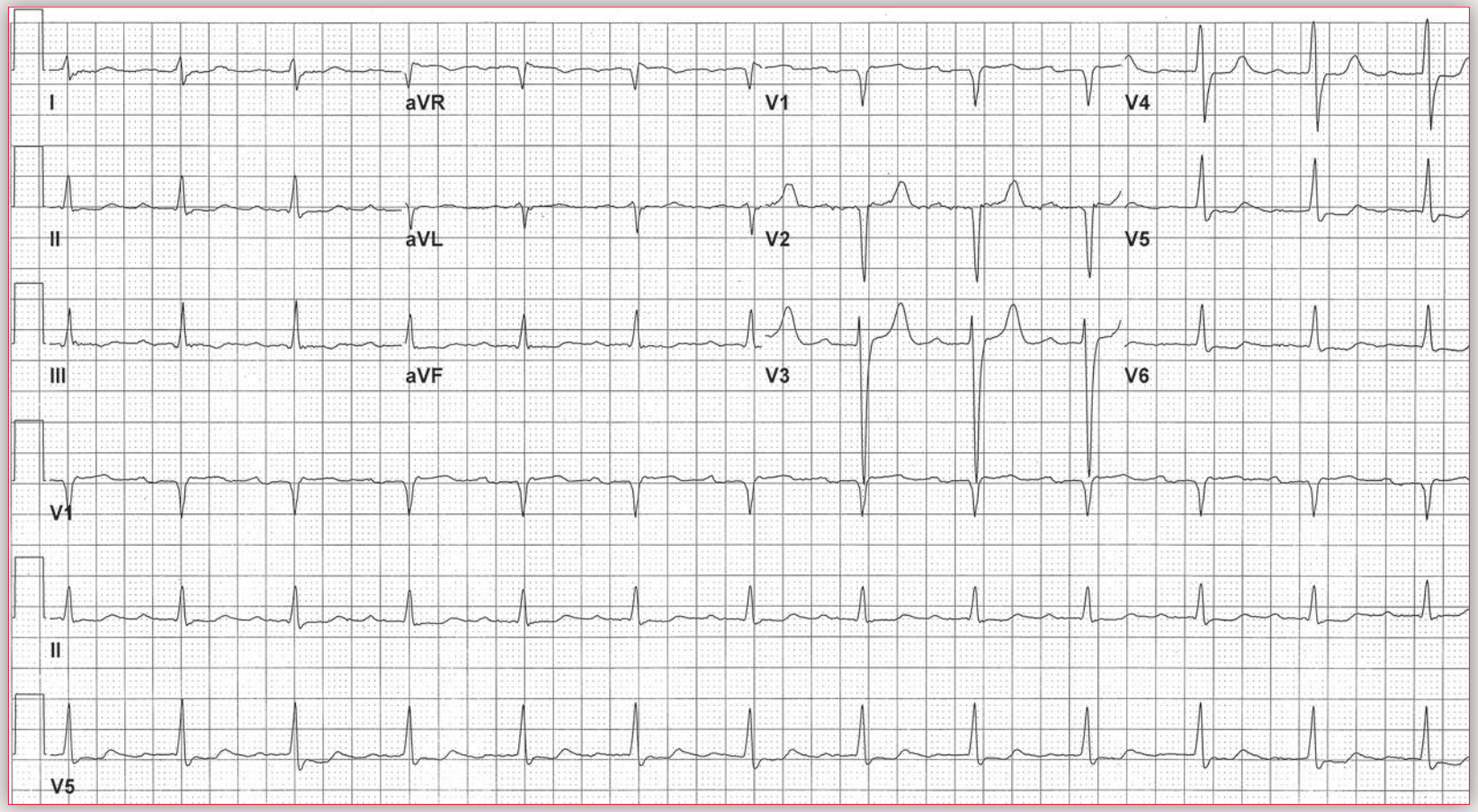


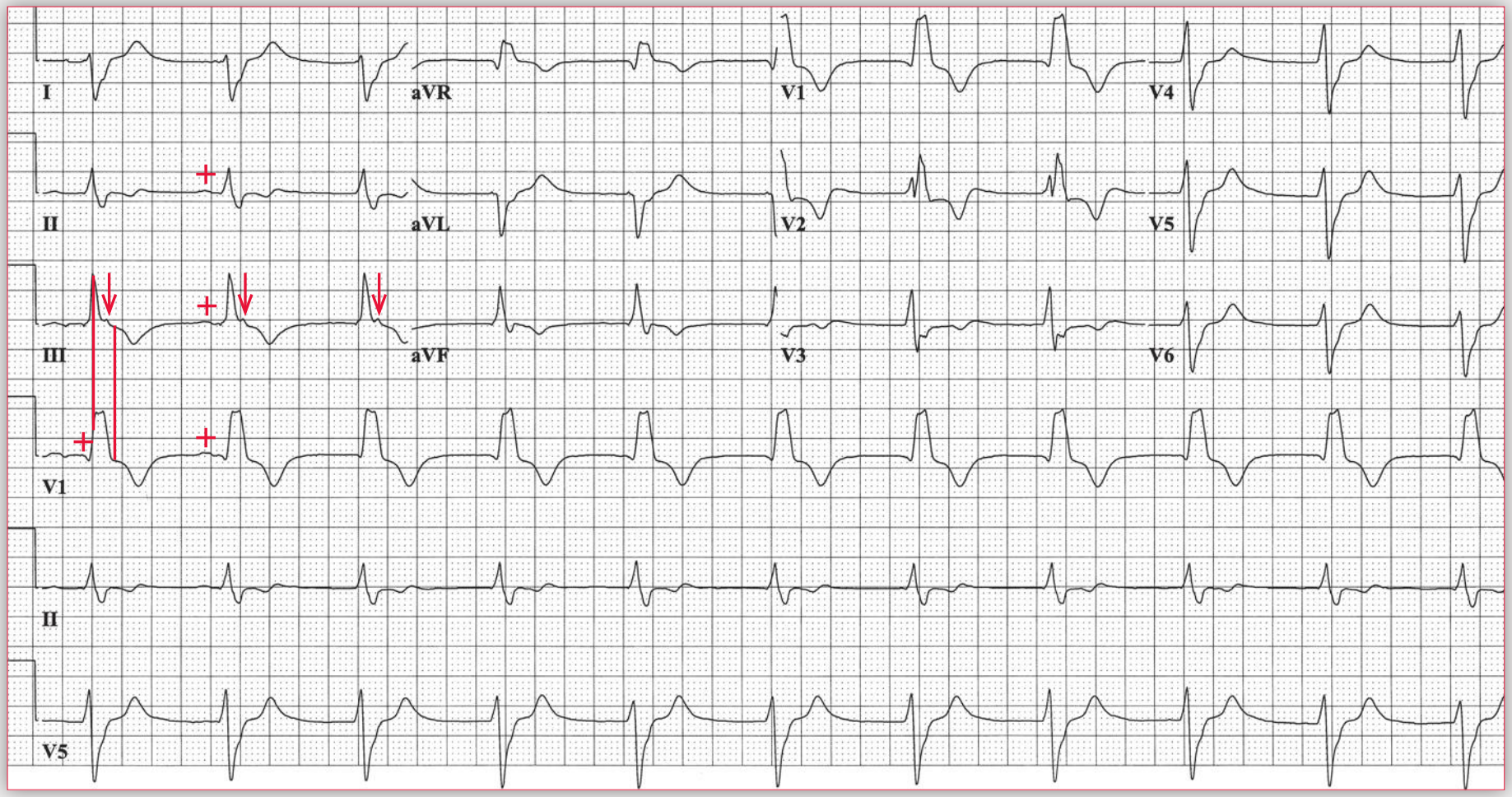
Practice Case 111

What rhythm abnormality is notable in ECG 111A?

What finding on the ECG is helpful in establishing the etiology of the rhythm disorder?

ECG 111B





ECG 111A Analysis: Idioventricular rhythm with AV dissociation

ECG 111A shows a regular rhythm at a rate of 64 bpm. There does not appear to be any obvious atrial activity. Although the small deflection seen in lead III (↓) is suggestive of a P wave, measuring the QRS width in lead I or V1 (‖) confirms that this waveform is actually part of the QRS complex. A P wave can be seen before the first and second QRS complexes (+) in lead V1 but not before any other QRS complex. This is consistent with the presence of AV dissociation, which is established by seeing a P wave before some, but not all, of the QRS complexes.

The QRS complexes have a right axis, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF) and an abnormal morphology that is not typical for either a right or left bundle branch block. This is an idioventricular rhythm, although it could also be considered a junctional rhythm with aberration (*ie*, right bundle branch block and left posterior fascicular block). However, the QRS complex does not have the typical morphology of a right bundle branch block and, more importantly, the presence of probable AV dissociation with a wide complex rhythm is consistent with a ventricular origin and makes a junctional rhythm less likely.

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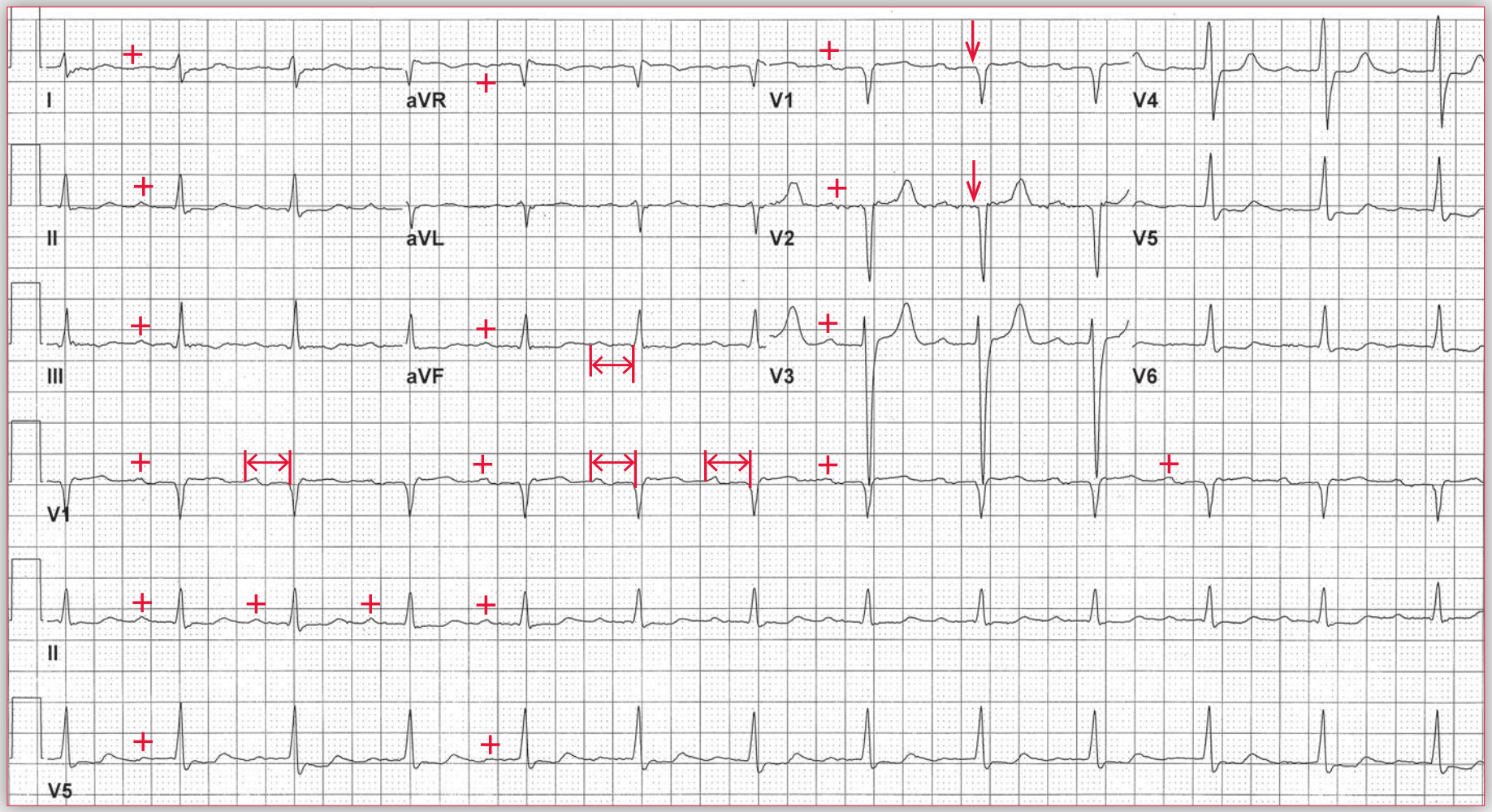


ECG 111B Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), old anteroseptal MI

ECG 111B shows a regular rhythm at a rate of 76 bpm. A P wave (+) is seen before each QRS complex with a stable but prolonged PR interval (↔) (0.28 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with first-degree AV block or prolonged AV conduction. The QRS complex axis is vertical, at about +90° (isoelectric QRS complex in lead I and positive QRS complex in lead aVF). The QRS complex duration is normal (0.10 sec). A QS complex in leads V1-V2 (↓) is diagnostic of an old anteroseptal myocardial infarction. The QT/QTc intervals are normal (390/440 msec).

Compared with the QRS complexes in **ECG 111A**, those in **ECG 111B** are normal during the faster rate of sinus rhythm. Therefore, **ECG 111A** does not show a junctional rhythm with aberrancy because the rate is slower yet the complexes are wider. As indicated, the QRS complexes in **ECG 111A** have a very abnormal morphology and AV dissociation is present, both features consistent with an idioventricular rhythm. ■

Notes

Practice Case 112

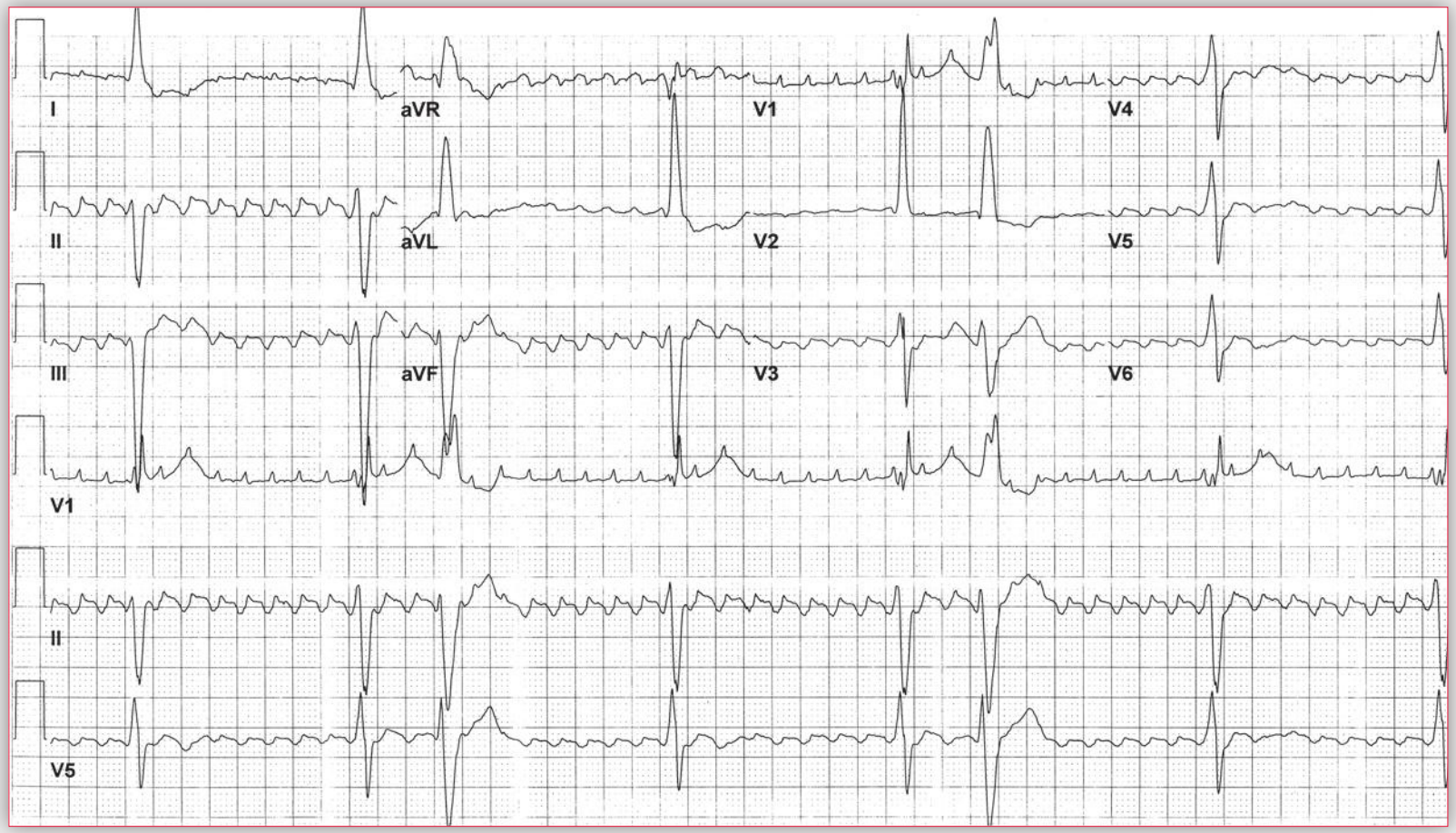
A 41-year-old woman with advanced familial hypertrophic cardiomyopathy is admitted to the hospital with syncope. The patient states that she felt lightheaded while preparing lunch at work and shortly thereafter found herself on the floor. Her colleagues called 911. The emergency medical technician (EMT) notes that the woman was conscious when they arrived at her workplace, and their mobile telemetry unit revealed atrial fibrillation with a slow heart rate.

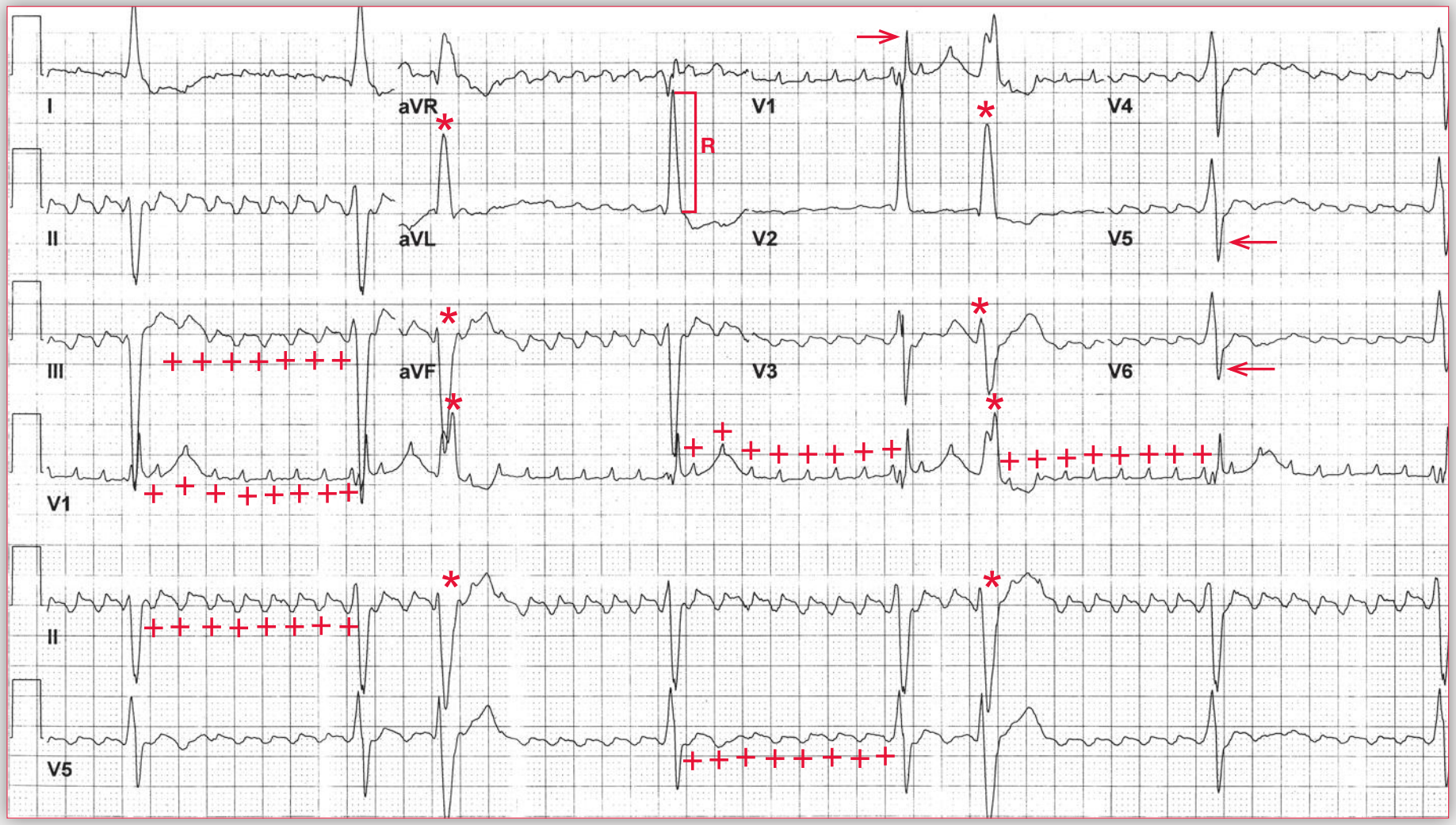
The woman is hemodynamically stable but has a heart rate of 40 bpm. Her cardiac exam is notable for an S4 gallop, but no murmur is appreciated. The admitting physician obtains an ECG.

What abnormalities are notable in the ECG?

Do you agree with the EMT's diagnosis?

What therapy would be appropriate?





ECG 112 Analysis: Atrial flutter with 8:1 conduction, premature ventricular complex, right bundle branch block, left anterior hemiblock, left ventricular hypertrophy

The rhythm is basically regular, but there are two premature complexes (third and sixth) (*) that are wide and abnormal with a morphology that differs from the others. These are premature ventricular complexes. All the other RR intervals are identical. Hence this is a regularly irregular rhythm at a rate of 38 bpm. Because there is a high degree of AV block (8:1), prominent flutter waves (+) can be seen at a regular rate of 300 bpm. The flutter waves are uniform in morphology, amplitude, and interval and have a typical undulating (saw-tooth) pattern. There is no isoelectric baseline between each flutter wave. The important characteristics of atrial flutter are a regular atrial rate of greater than 260 bpm with uniformity of all the waveforms and a consistent undulation of the waveforms without an isoelectric baseline between them. This is because atrial flutter is the result of a single reentrant circuit (in the right atrium) and consistent and continuous electrical activity is generated as a result.

The QRS complexes are wide (0.16 sec) and have a right bundle branch block morphology (RSR′ in lead V1 [→] and broad S wave in leads V5-V6 [←]). The axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). There are two etiologies for an extreme left axis, *ie*, an old inferior wall myocardial infarction with initial deep Q waves in leads II and aVF or a left anterior fascicular block in which there is an rS morphology of the QRS complex in leads II and aVF. Hence, this is a left anterior fascicular block. This conduction abnormality (*ie*, right bundle branch block and left anterior fascicular block) is termed bifascicular block. The R-wave amplitude in lead aVL is 20 mm ( ]), meeting criteria for left ventricular hypertrophy (*ie*, R-wave amplitude in lead aVL > 18 mm in the presence of a left axis). The QT/QTc intervals are normal (600/480 msec and 540/420 msec when corrected for the prolonged QRS complex).

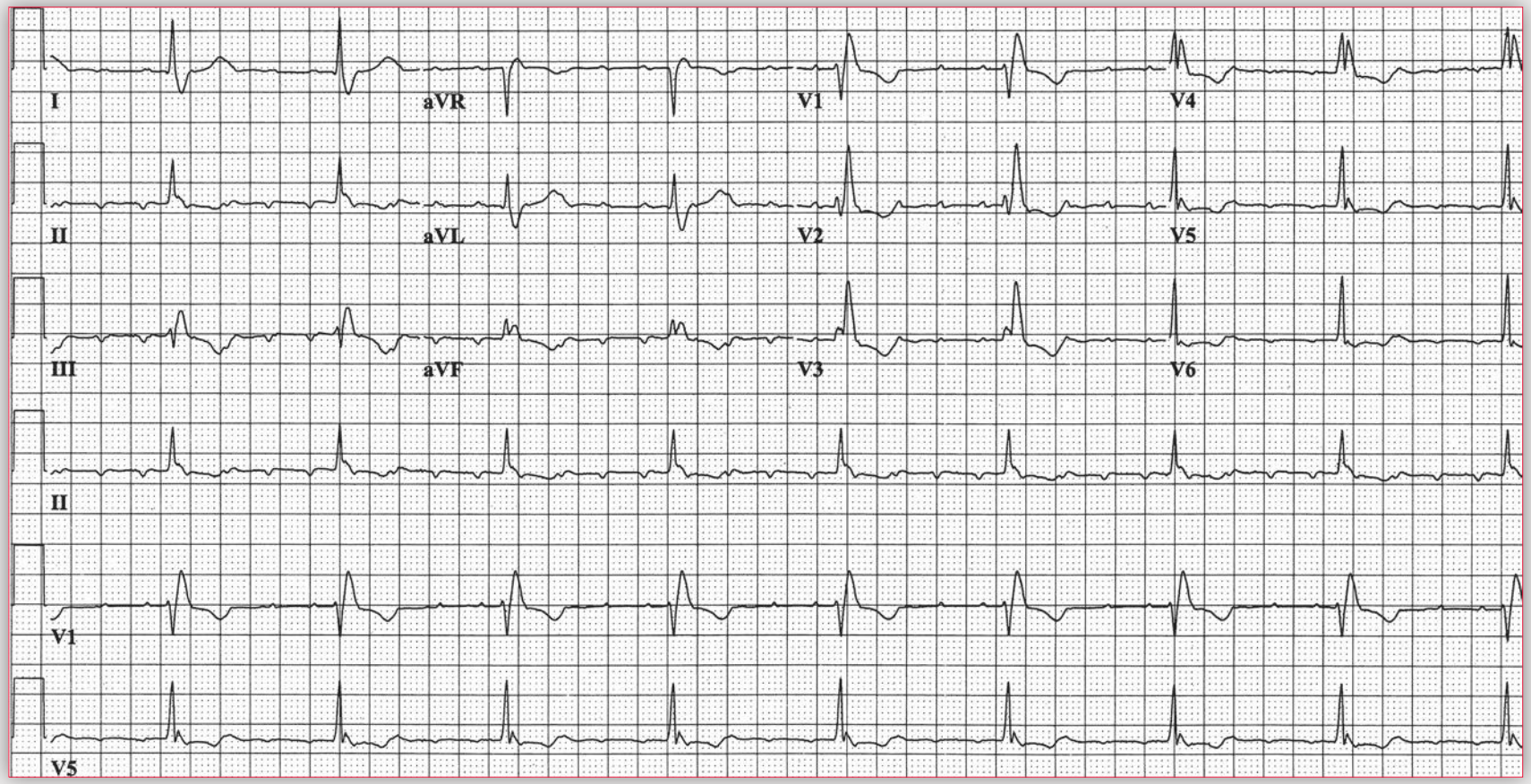
The patient has evidence of significant conduction system disease with high-degree AV block as well as bifascicular block. The conduction system disease is likely the result of the underlying cardiomyopathy. The presence of advanced conduction abnormalities suggests complete heart block as a cause of syncope. The treatment of choice for complete heart block and symptomatic bradycardia is placement of a pacemaker. However, another cause for syncope in patients with hypertrophic cardiomyopathy is ventricular tachyarrhythmia. Hence the etiology for the syncope should be further evaluated and established before pursuing a specific therapy. ■

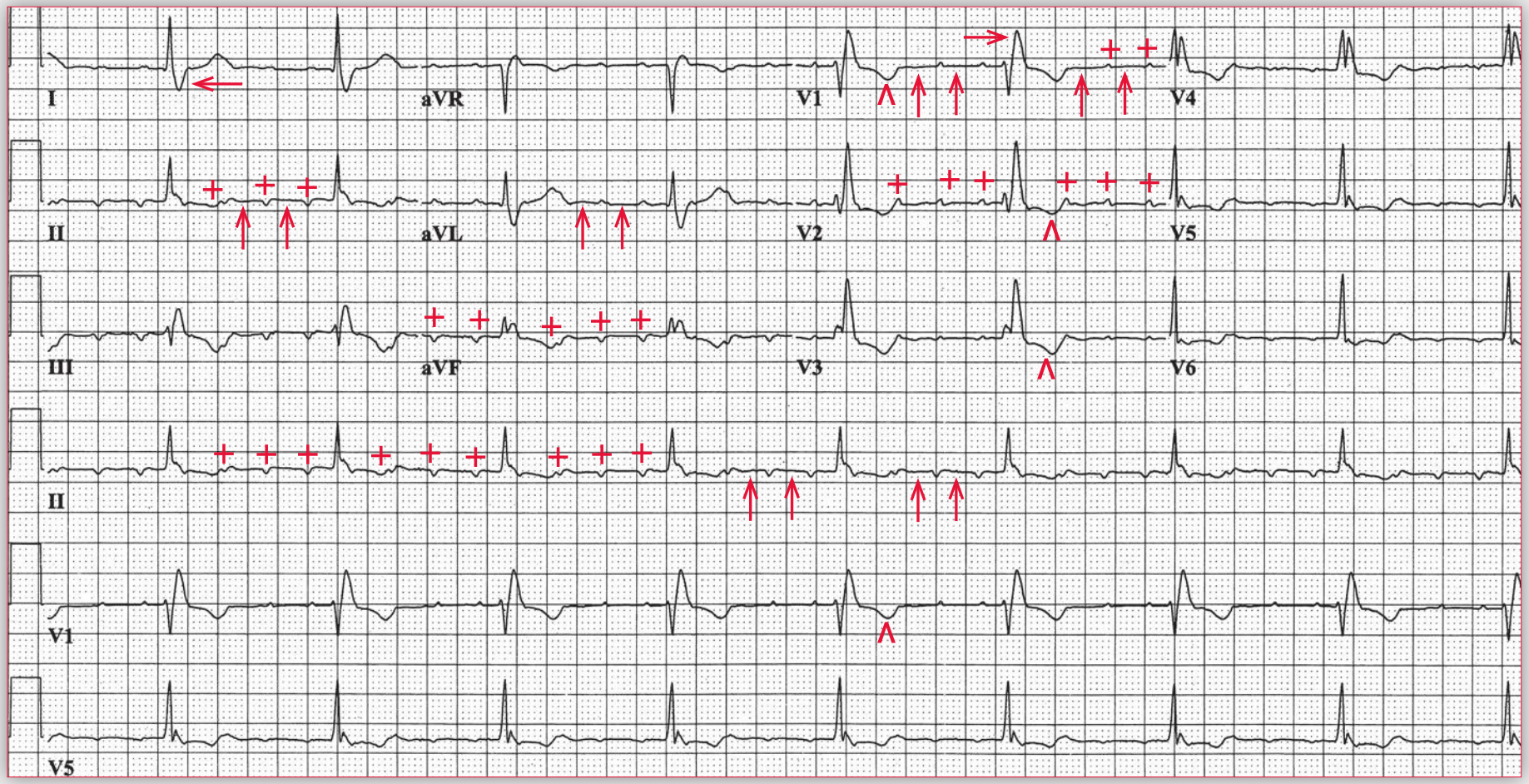
Notes

Practice Case 113

A 28-year-old man with severe asthma is admitted to the hospital with status asmaticus. He is on theophylline at home and is being treated in the hospital with nebulized β-agonists. An ECG is obtained.

What does the ECG show?





ECG 113 Analysis: Atrial tachycardia with 4:1 conduction, nonspecific T-wave abnormalities

There is a regular rhythm at a rate of 54 bpm. Noted is an atrial rate of 220 bpm. The P waves (+) are distinct, and there is an isoelectric baseline (↑) between each P wave. The P waves are negative (inverted) in leads II, aVF, and V4-V6. This is, therefore, atrial tachycardia with 4:1 AV conduction. Atrial tachycardias can be precipitated by pulmonary disease or by sympathomimetic agents such as theophylline or β-agonists. When AV conduction is present, the PR interval is constant (0.20 sec).

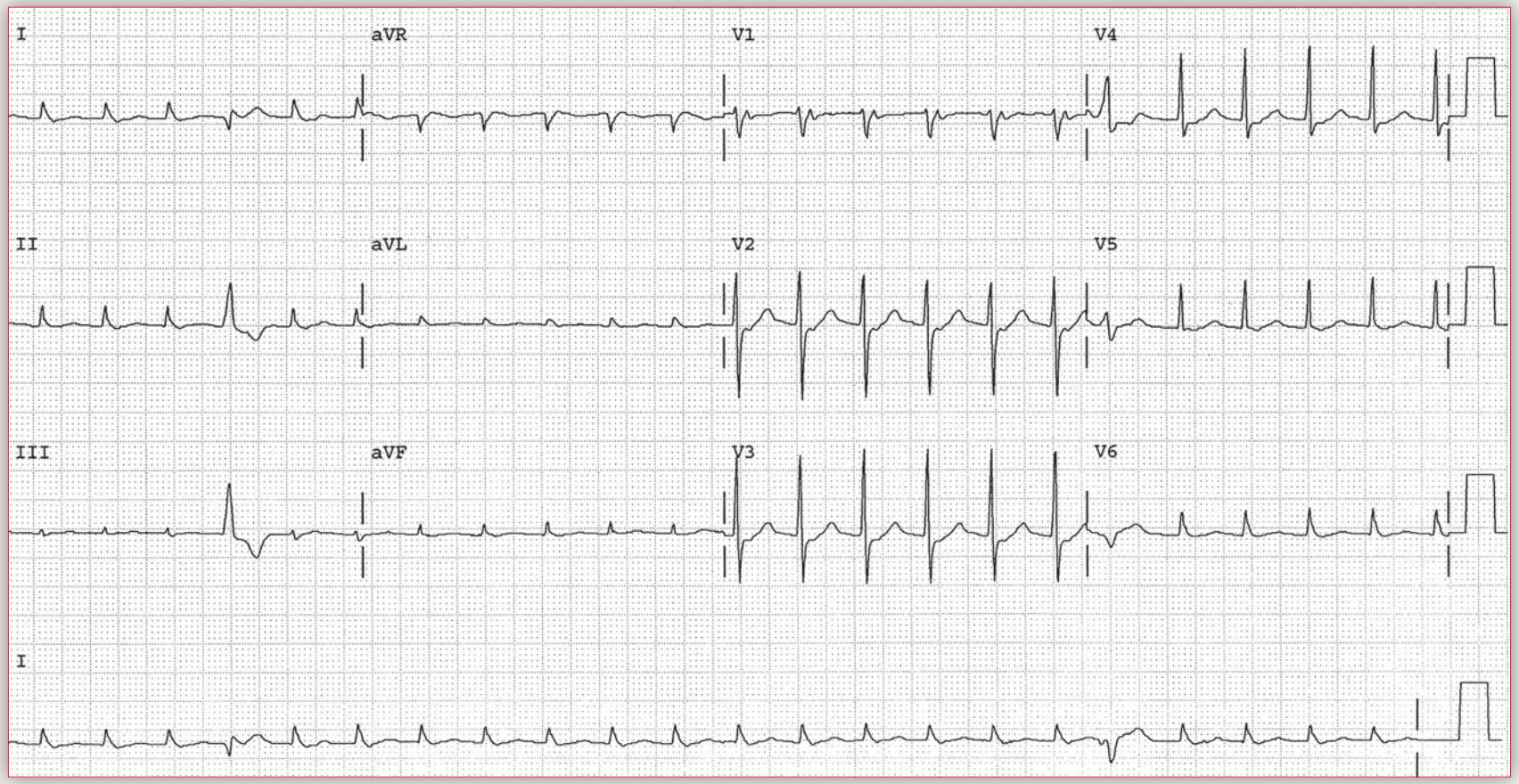
The QRS complexes are regular at a rate of 54 bpm. The QRS interval is prolonged (0.16 sec), and the QRS complex morphology is typical for right bundle branch block (RBBB; RSR′ morphology in lead V1 [→] and broad S waves in lead I [←]). The QT/QTc intervals are normal (440/420 msec and 380/360 msec when accounting for the prolonged QRS interval). T-wave inversions noted in leads V1-V3 (^) are secondary to the RBBB. There are also T-wave inversions in leads II, III, and aVF; these are primary but nonspecific.

The occurrence of atrial tachycardia is likely the result of excessive sympathomimetic agents used for treatment of asthma. What is unusual is the presence of 4:1 AV conduction. Given the presence of sympathomimetic agents, which should enhance AV nodal conduction, the high-degree AV block likely indicates the presence of intrinsic AV nodal disease in association with the presence of intraventricular conduction disease (*ie*, RBBB). ■

Practice Case 114

A 44-year-old woman with a long history of "palpitations" without a confirmed diagnosis presents to your primary care clinic with accelerating palpitations. She states that she is having salvos of "rapid heartbeats" multiple times a day. She denies

ECG 114A

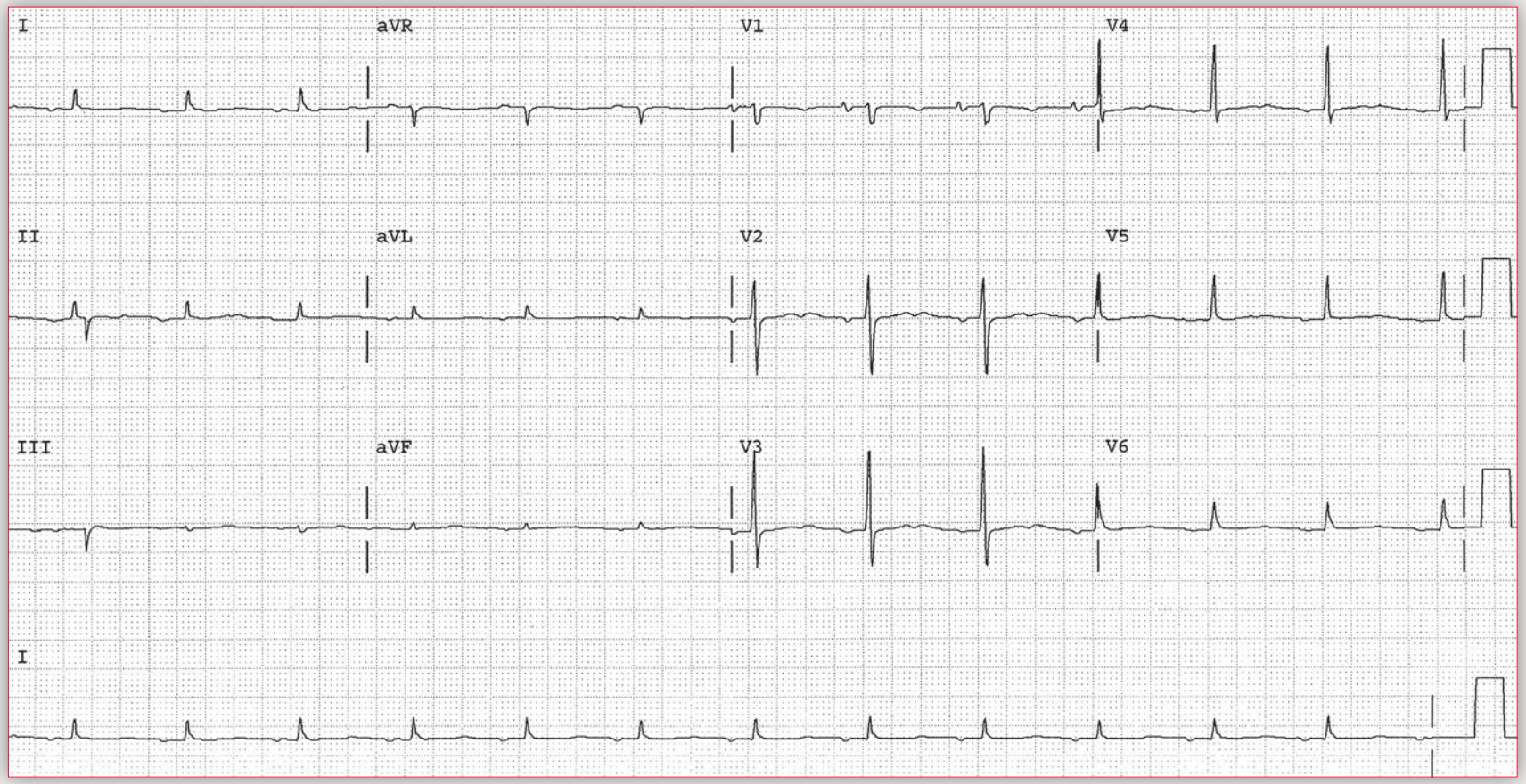


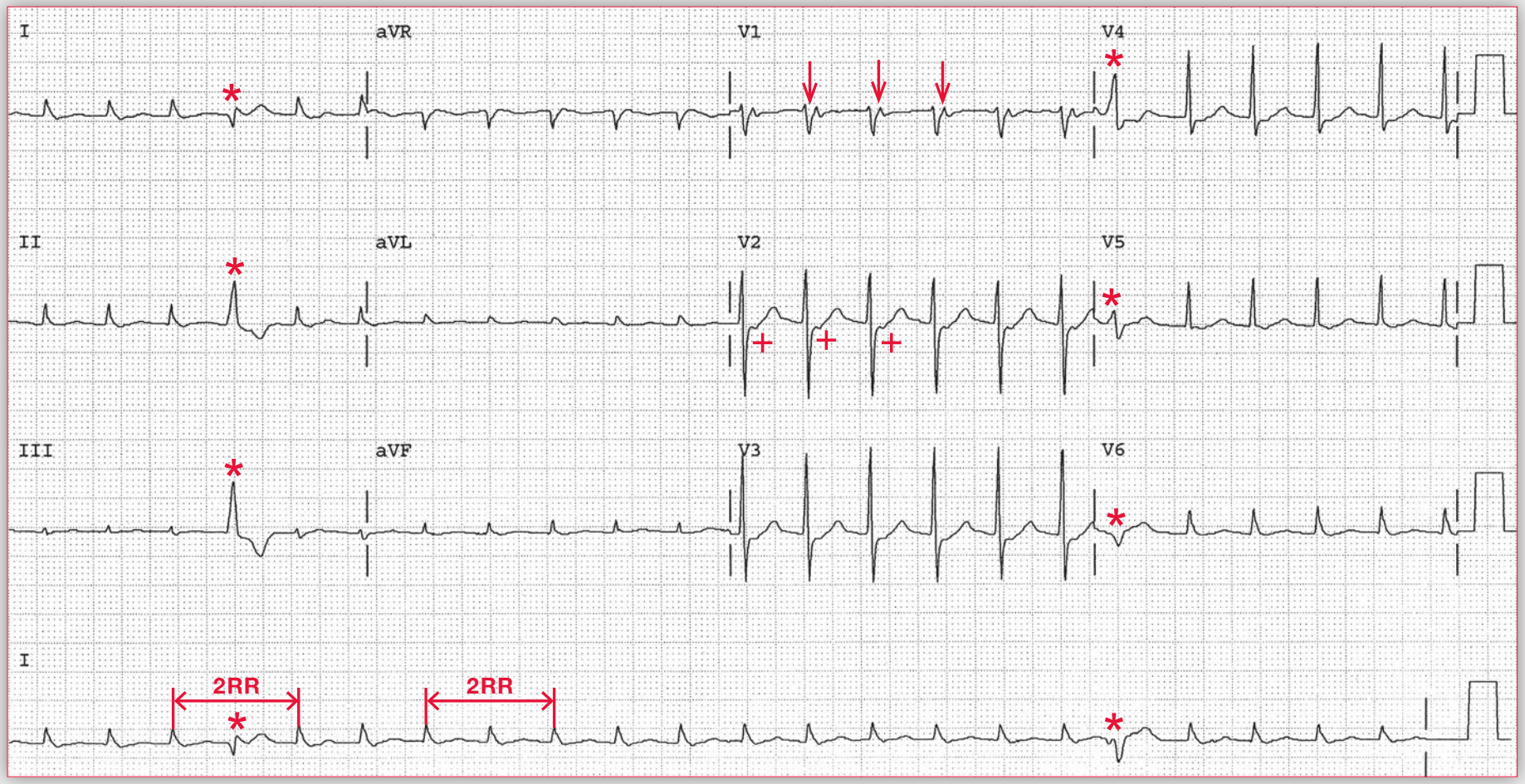
any associated symptoms but says the sensation itself is extremely distressing. On exam, you note for the first time that she has a rapid radial pulse. You quickly obtain and ECG (114A) and retrieve a prior ECG (114B) for comparison.

What abnormalities are notable on ECG 114A?

In comparing the two tracings, what disorder of cardiac conduction is suggested?

ECG 114B





ECG 114A Analysis: Low-voltage limb leads, AV nodal reentrant tachycardia, premature ventricular complexes

In **ECG 114A** there is a regular rhythm at a rate of 140 bpm. No P waves are seen before or after any of the QRS complexes. The QRS complex duration is normal (0.08 sec), as are the QT/QTc intervals (290/440 msec). The QRS complex is of low voltage in the limb leads (amplitude < 5 mm in each limb lead), but the morphology is normal with a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). However, there is what appears to be an R′ waveform in lead V1 (↓) at the very end of the QRS complex suggesting a right ventricular conduction delay, although no S waves are seen in leads I and V5-V6. Hence it is possible that this represents a retrograde P wave superimposed on the end of the QRS complex, which is also suggested by the presence of a small positive waveform at the end of the QRS complex in leads V2-V3 (+). However, it is not a distinct P wave. Hence this is a no RP tachycardia, the most common etiology for which is AV nodal reentrant tachycardia.

In addition, there are two early, wide complexes that are premature ventricular complexes (*). They do not alter the underlying rhythm, and the RR interval surrounding these premature ventricular complexes is equivalent to two RR intervals (↔).

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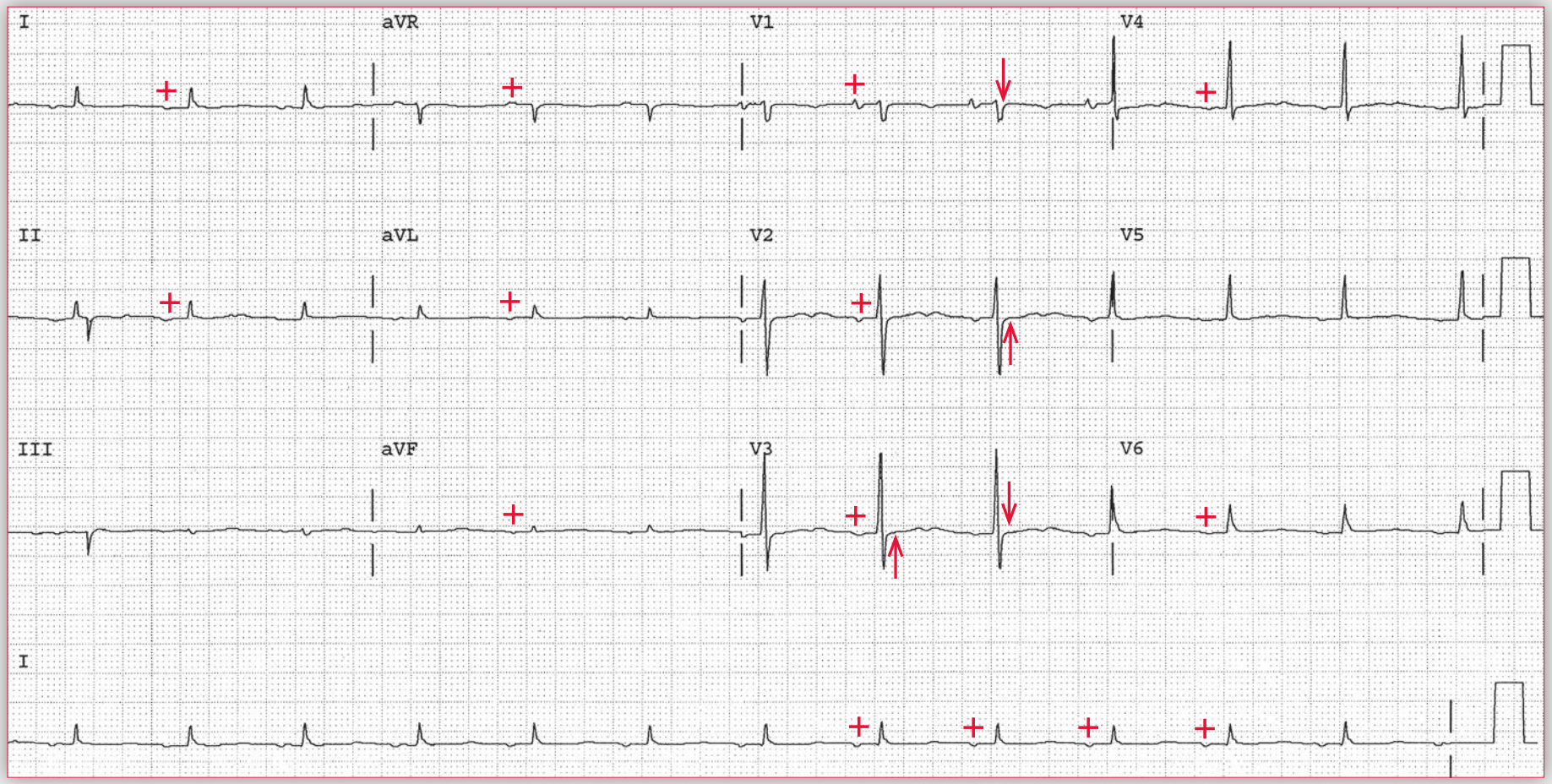


ECG 114B Analysis: Low-voltage limb leads, atrial rhythm, diffuse nonspecific T-wave abnormalities

ECG 114B shows a regular rhythm at a rate of 76 bpm. The QRS complex morphology, duration, and axis are the same as in **ECG 114A**. The QT/QTc intervals are normal (380/430 msec), However, the R′ waveform in lead V1 of **ECG 114A** is not seen (↓), nor are the small positive waveforms in leads V2-V3 (↑), confirming that the waveforms seen in **ECG 114A** were indeed retrograde P waves. There is a P wave (**+**) in front of each QRS complex with a stable PR interval; however, the P wave is negative in leads I, II, aVF, and V4-V6. Hence this is an atrial rhythm. ■

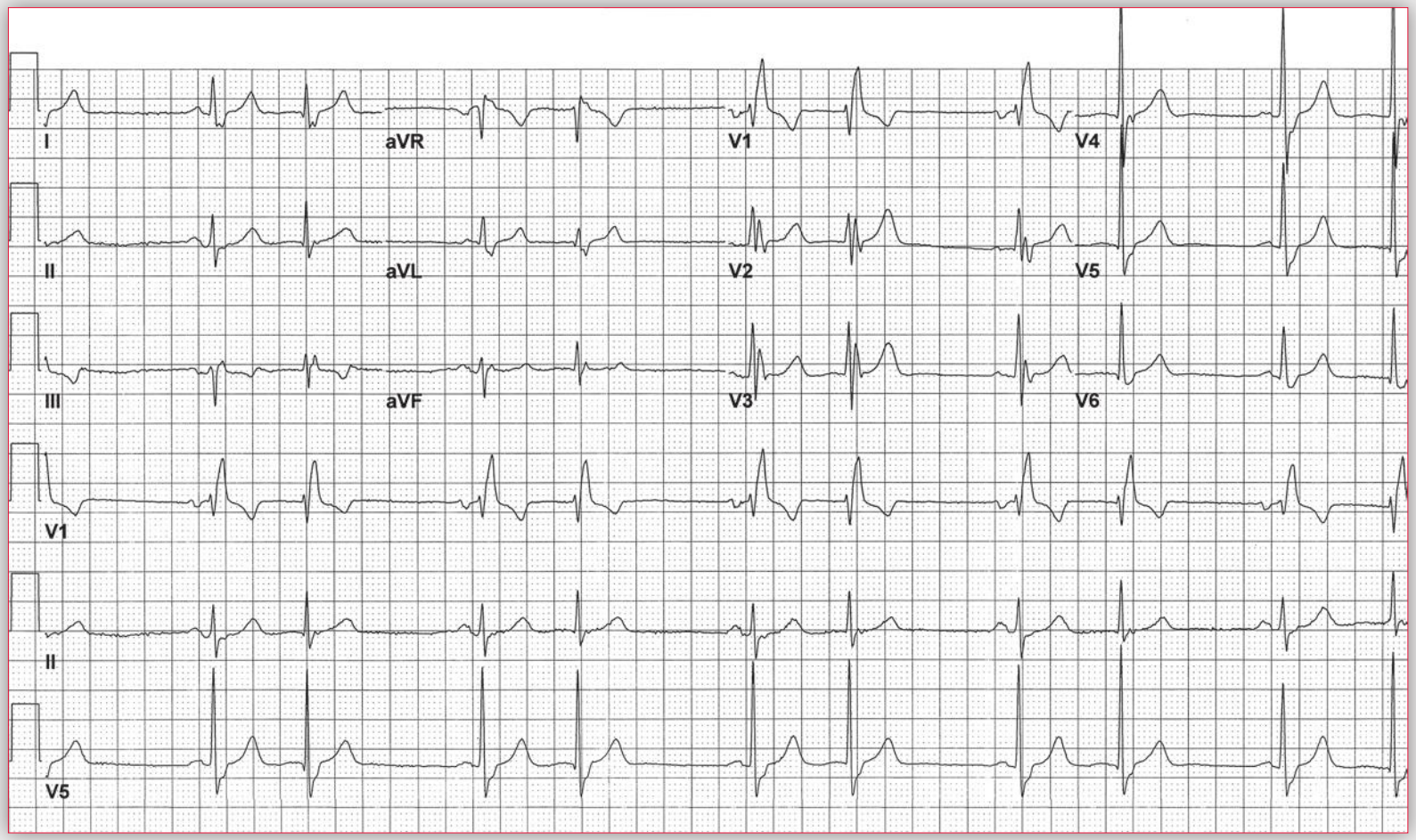
Notes

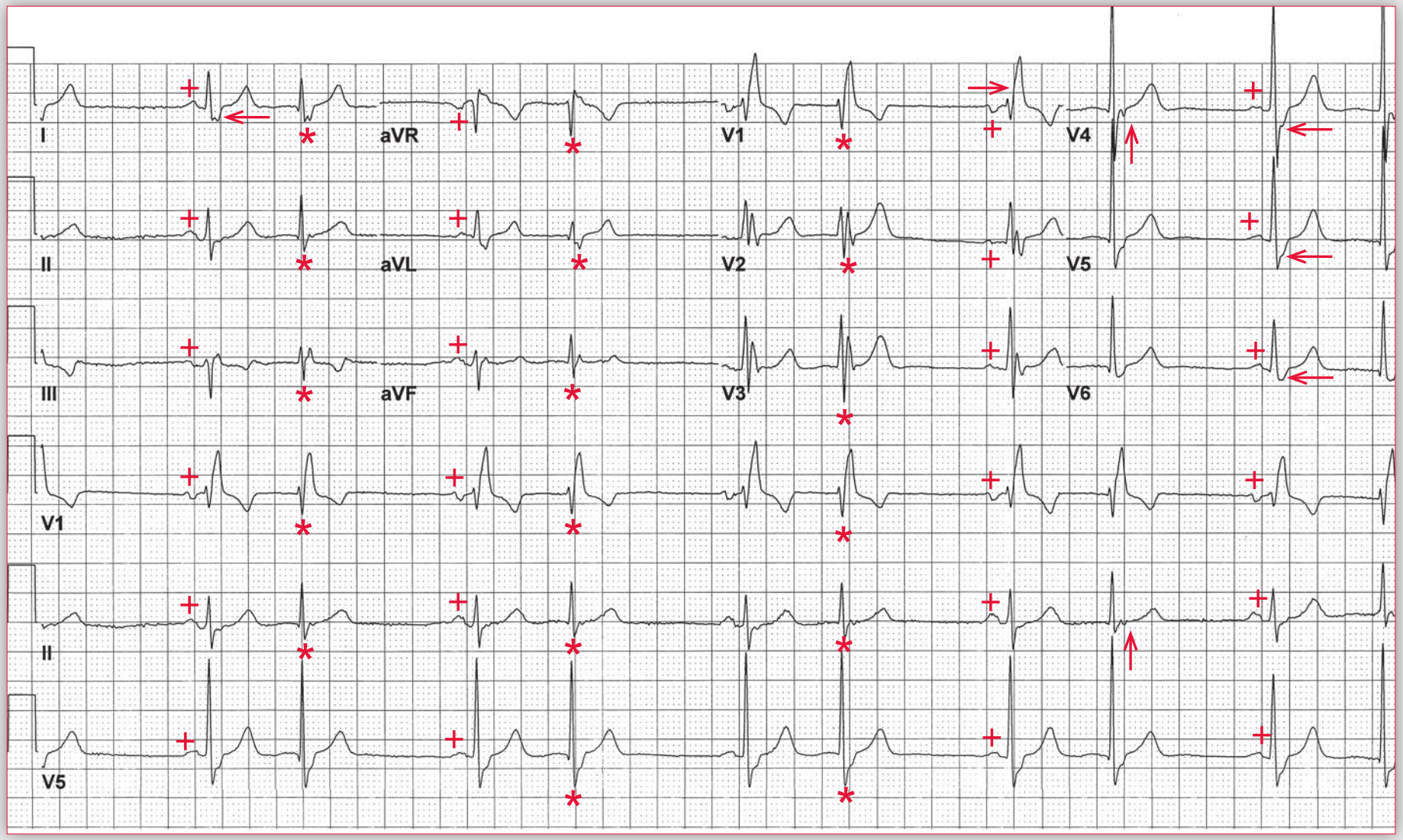
Practice Case 115

A 28-year-old man in the medical intensive care unit is critically ill with septicemia of unknown source. He is undergoing pulmonary artery catheter insertion to assist with management of depressed left ventricular function noted on a surface echocardiogram. During the bedside procedure, the assisting house officer notes a change in the patient's QRS complex morphology and an alteration in his cardiac rhythm on telemetric monitoring. The procedure is aborted, and an ECG is obtained.

What disturbances of cardiac conduction are notable on the tracing?

What abnormalities may be related to the catheter insertion?





ECG 115 Analysis: Normal sinus rhythm, left axis, right bundle branch block, premature junctional complexes in bigeminal pattern (junctional bigeminy)

There is a pattern of group beating, with two QRS complexes and a pause. The rhythm is, therefore, regularly irregular. The first QRS complexes are preceded by a P wave (+) with a constant PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. The QRS complex duration is increased (0.16 sec), and it has an RSR′ morphology in lead V1 (→) and a broad S wave in leads I and V4-V6 (←), diagnostic for a right bundle branch block (RBBB). There is a physiologic left axis of the sinus complex (positive QRS complex in leads I and II and negative QRS complex in lead aVF). Following each sinus complex is an early QRS complex (*) with the same RBBB morphology. There is, however, no P wave prior to this complex and hence these are premature junctional complexes (PJCs) in a bigeminal pattern. There are small waveforms after the PJCs (↑), seen in leads II and V4, which are likely retrograde P waves.

Although the PJC has the same morphology and duration as the sinus complex, it has a normal axis (positive QRS complex in leads I, II, and aVF) in contrast to the sinus complex. In addition, the QRS amplitude of the PJC is slightly different from that of the sinus complex. It is very common for junctional complexes to have a slightly different amplitude or axis compared with sinus complexes. This is due to the fact that the junctional complex, which originates from an ectopic focus within the AV junction, penetrates the His-Purkinje system, which is a series of tracts at a slightly different location compared with the sinus impulse going through the AV node. As the location of conduction through the His-Purkinje system differs from that of a sinus impulse, the QRS complex originating in the junction may have a slightly different amplitude or axis compared with the sinus complex.

Right heart catheter insertions may be associated with transient RBBB in 5% of patients due to mechanical irritation of the right bundle, which has a superficial location on the right side of the intraventricular septum. The junctional arrhythmia may also be caused by the right heart catheter, which goes through the tricuspid annulus, and mechanical irritation may cause PJCs. However, this arrhythmia may also be related to the underlying cardiomyopathy or the patient's critical illness. ■

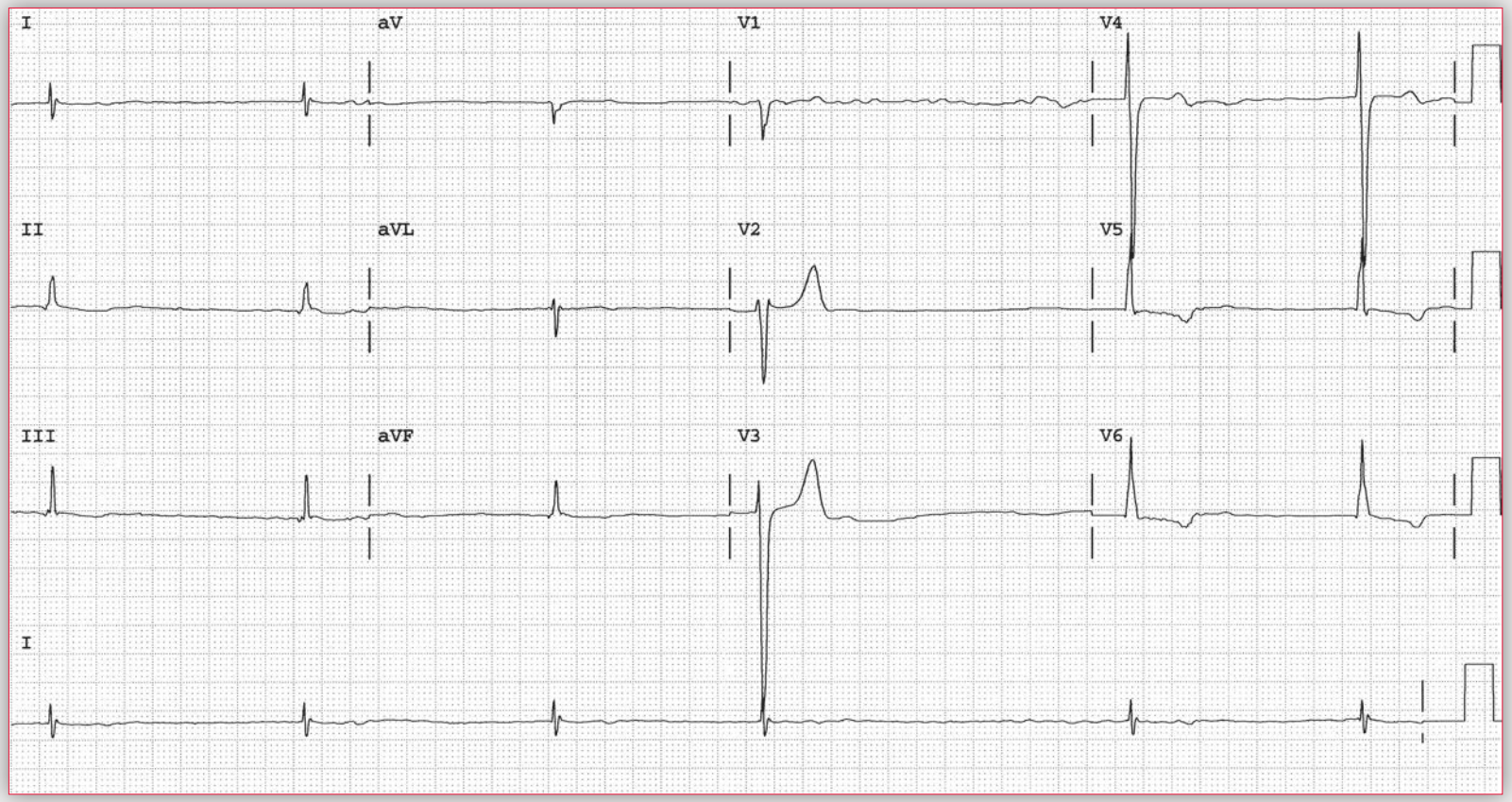
Notes

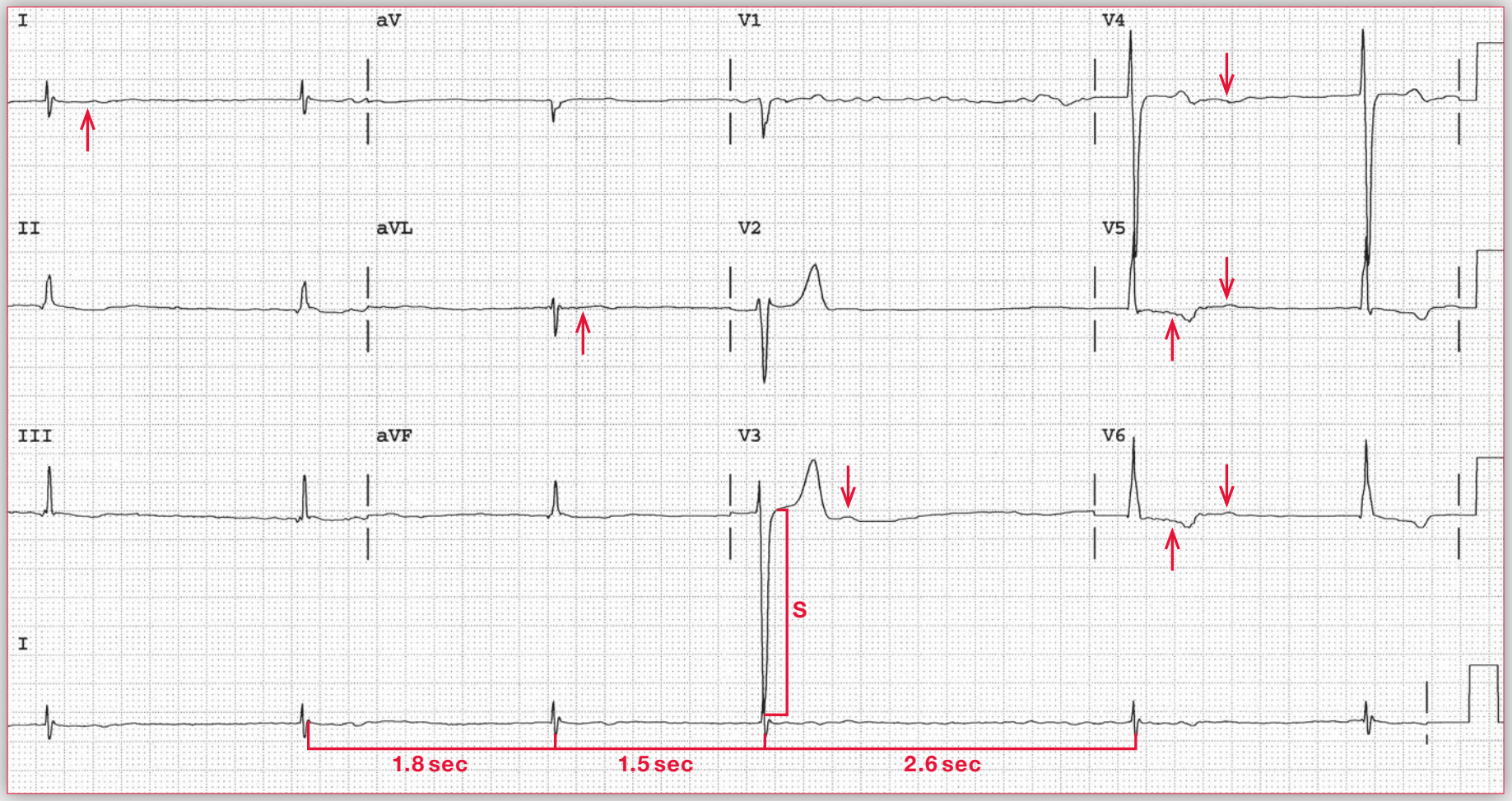
Practice Case 116

An 86-year-old man is brought to the emergency department after being recovered from a scene of a motor vehicle accident. He was found unconscious by emergency personnel, apparently having lost consciousness while driving and subsequently losing control of his automobile.

On presentation, he is unconscious in a cervical spine immobilization device. His heart rate is 36 bpm, and his blood pressure is 76/40 mm Hg. The primary survey reveals extremity abrasions but no signs of blunt force trauma or gross limb fracture. A surface 12-lead ECG is obtained before β-agonist infusion is begun for therapy of both hypotension and bradycardia.

What finding on the ECG explains the patient's presentation?





ECG 116 Analysis: Atrial fibrillation with slow ventricular response, left ventricular hypertrophy, ST-T wave abnormalities

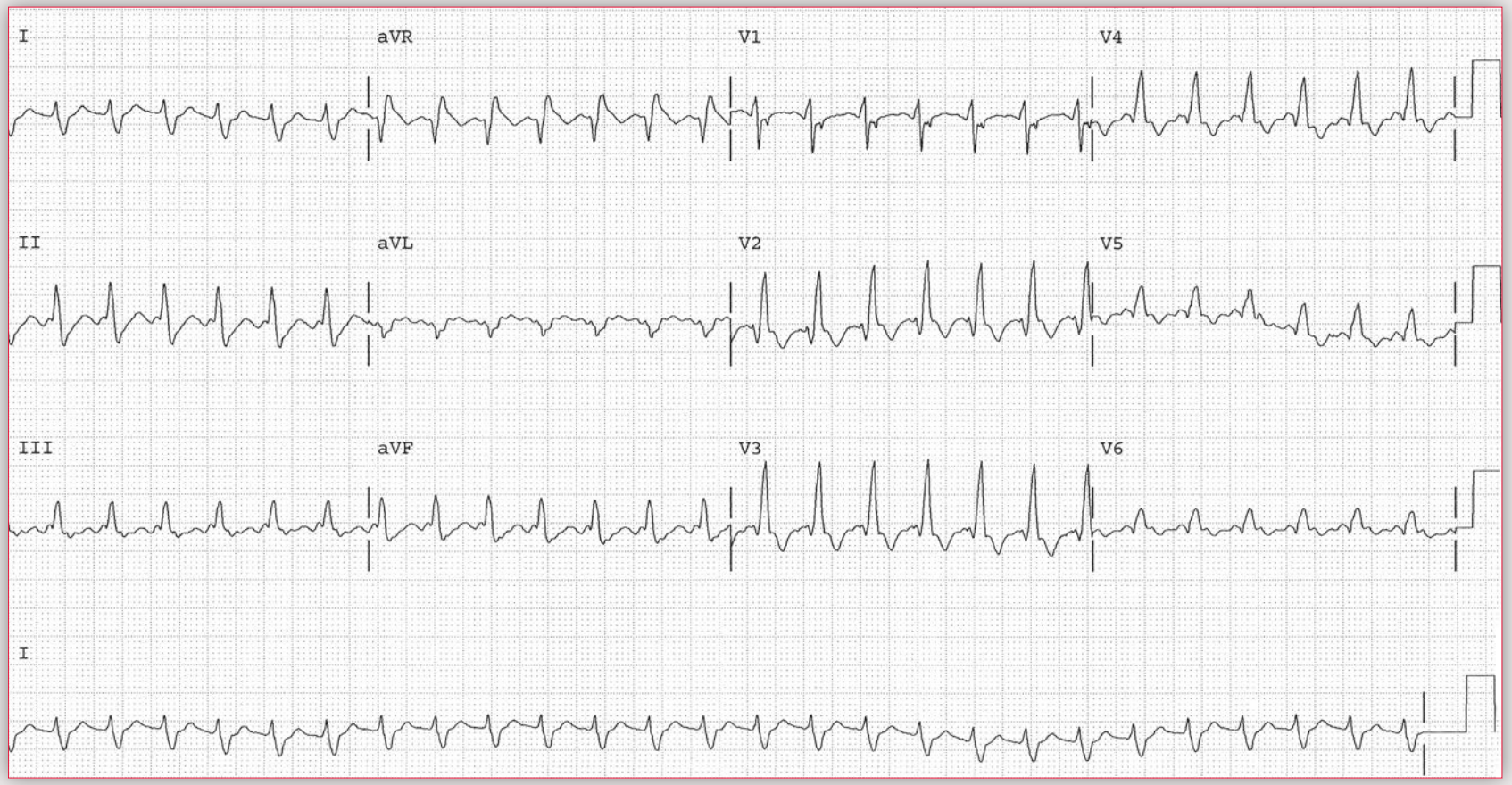
The rhythm is irregularly irregular at a rate of 36 bpm. There are only three supraventricular rhythms that are irregularly irregular: (1) sinus arrhythmia in which there is one P-wave morphology and PR interval, (2) multifocal atrial rhythm (wandering atrial pacemaker) with a rate less than 100 bpm or multifocal atrial tachycardia with rate greater than 100 bpm in which there are three or more different P-wave morphologies and no P-wave morphology is dominant, or (3) atrial fibrillation, in which there is no organized atrial activity but there are fibrillatory waves. There is no obvious atrial activity, and hence this is atrial fibrillation with a very slow ventricular response rate. This may be the result of intrinsic AV nodal disease, very high vagal tone, or excessive effect from AV nodal blocking agents. The QRS complexes are of normal duration (0.08 sec) and have a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (500/390 msec). There is an increased QRS voltage, particularly in leads V3-V4 (S-wave depth in lead V3 = 35 mm [[ ]). This is diagnostic for left ventricular hypertrophy (*ie*, R wave or S wave in any precordial lead ≥25 mm). There are ST-T wave abnormalities (↑) associated with the hypertrophy, especially in leads V5-V6. Also noted are small U waves in leads V3-V6 (↓). It is possible that hypokalemia is present, although prominent U waves are also seen when bradycardia is present.

The etiology for the loss of consciousness is not certain. Although the patient has atrial fibrillation with a slow ventricular rate, this is associated with a measurable blood pressure (76/40 mm Hg). Therefore, the slow ventricular response rate is not resulting in major hemodynamic compromise and is not likely to be the cause of the loss of consciousness. It is possible that there is some intracerebral process (either the reason for the loss of consciousness or the result of head trauma) that accounts for the hypotension and slow ventricular rate. The slow ventricular response rate may have preceded the accident, which would suggest that there may have been an even slower rate or possibly long periods of asystole that accounted for the loss of consciousness. It is also possible that this was the result of a vasovagal episode and that the persistent loss of consciousness reflects head trauma. ■

Practice Case 117

A 72-year-old man with known atrial fibrillation on long-term warfarin therapy is electively admitted for direct-current cardioversion. He is administered propofol anesthesia and undergoes transesophageal echocardiography, which documents the absence of left atrial or appendage thrombus.

ECG 117A



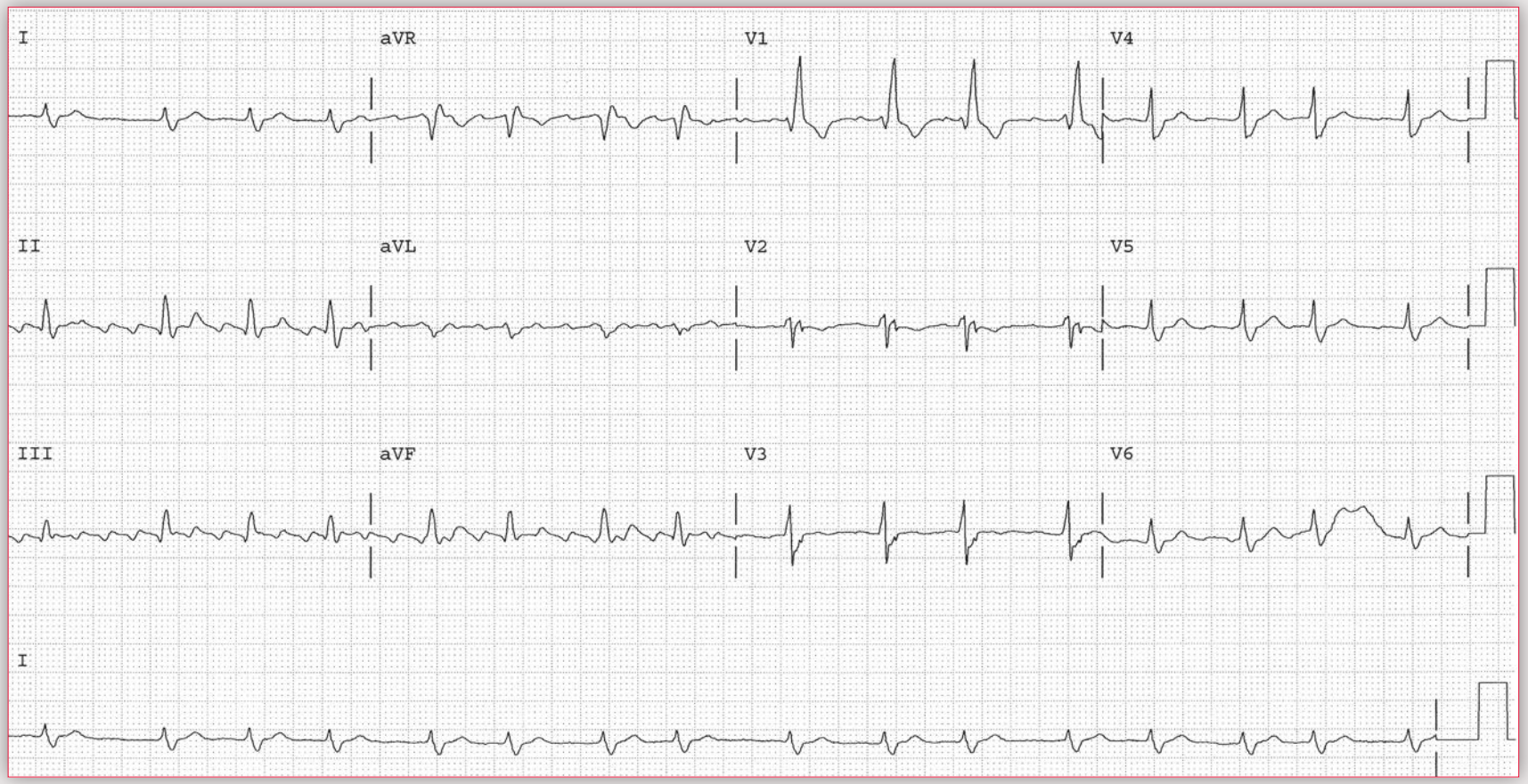
Practice Case 117

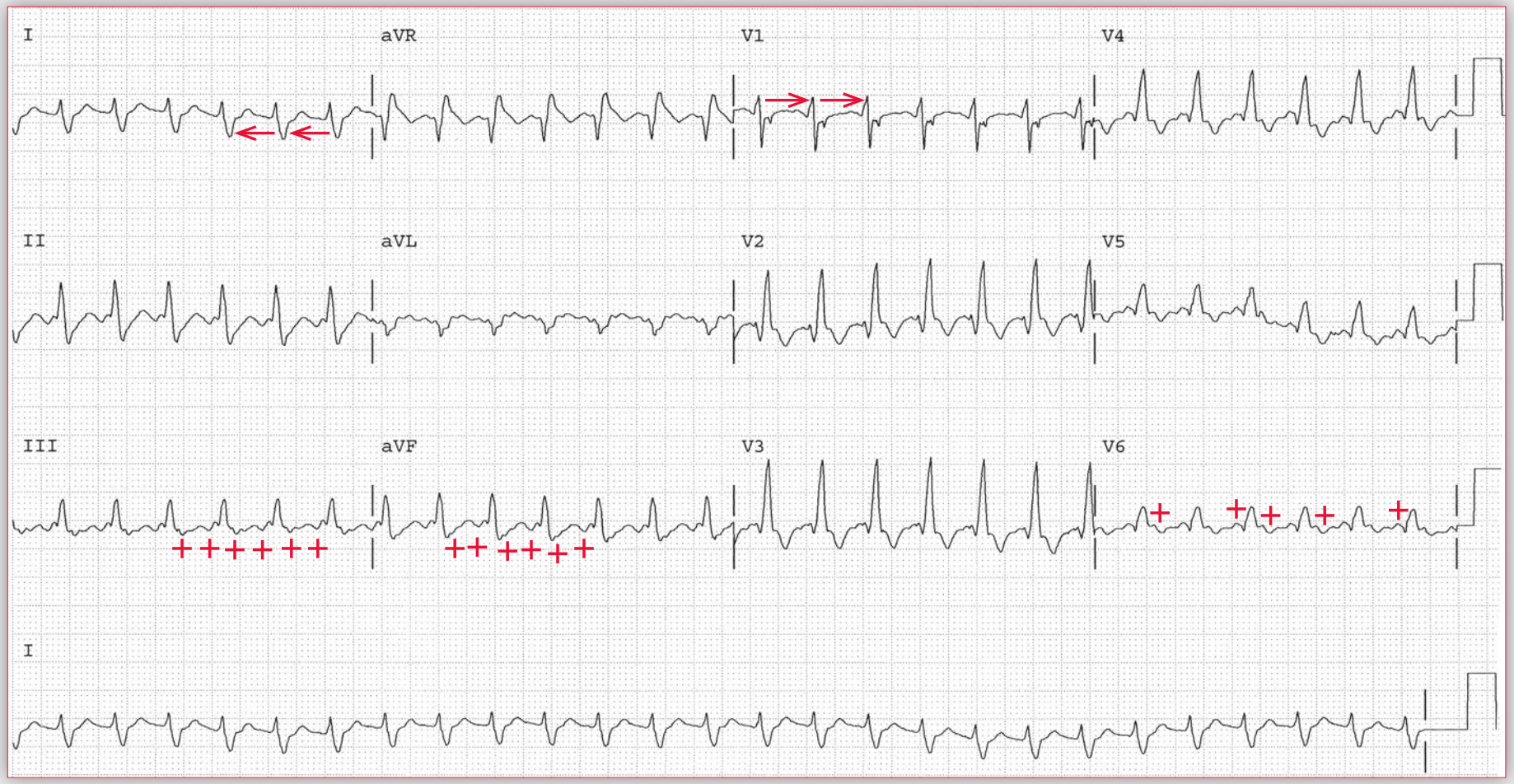
After an initial 50-J discharge, his heart rate is noted to be rapid. An ECG is obtained (ECG 117A). The patient is given another dose of propofol and undergoes a second discharge at 100 J. Upon seeing the tracing after the second discharge (ECG 117B), the electrophysiology fellow declares, "Well, he is back in atrial fibrillation."

What arrhythmias are notable on the two ECGs?

Do you agree with the fellow's interpretation?

ECG 117B





ECG 117A Analysis: Atrial flutter with 2:1 conduction

In **ECG 117A** the rhythm is regular at a rate of 160 bpm. The QRS complex duration is prolonged (0.14 sec). There is a broad S wave in lead I (←) and a tall R wave in lead V2 (→), a pattern that is characteristic of, but not definitive for, right bundle branch block as lead V1 does not have an RSR′ pattern. This may be the result of lead malposition. The axis is normal, about 0° (biphasic QRS complex in lead I and positive QRS complex in lead aVF). The QT/QTc intervals appear to be slightly prolonged (280/460); however, when the prolonged QRS complex duration is considered, these intervals are normal (240/380 msec).

No obvious atrial waveforms can be seen in any lead, although notches or bumps before and after the QRS complex are seen in leads III and aVF as well as leads V5-V6 (+), suggestive of atrial activity. These notches are regular at a rate of 320 bpm, suggesting that the rhythm is atrial flutter with 2:1 AV conduction. It should be noted that the negative waveform after the QRS complex in lead V1 is not a P wave but rather is part of the QRS complex, which can be established by measuring the maximal QRS complex duration (*eg*, in lead V2) and comparing this QRS complex duration with that in lead V1.

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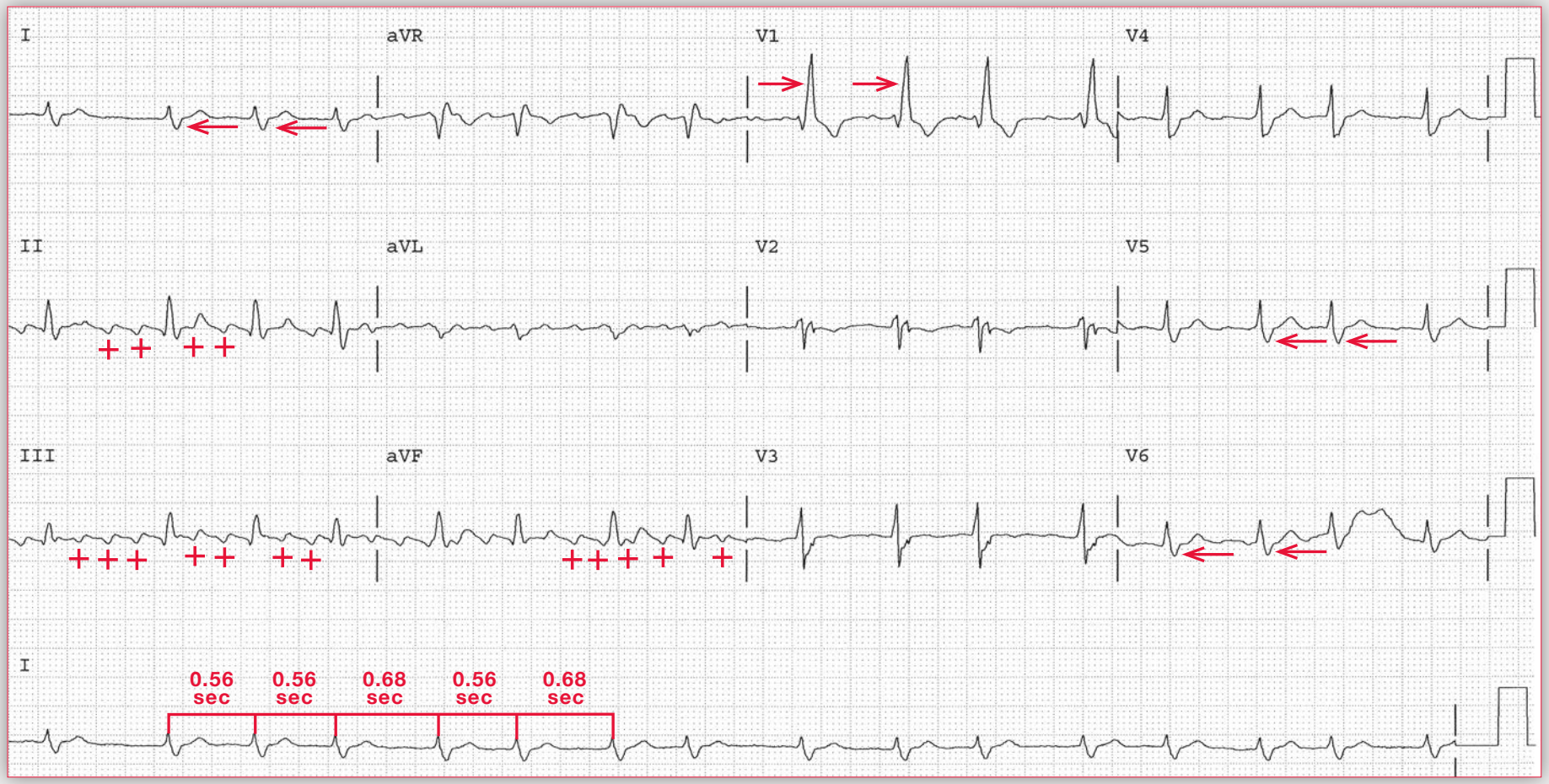


ECG 117B Analysis: Typical atrial flutter with variable AV block or conduction

The rhythm in **ECG 117B** is irregular at an average rate of 96 bpm. Although irregular, many of the RR intervals are the same and hence this rhythm is regularly irregular. The QRS complex morphology is now typical for a right bundle branch block, with an S wave in leads I and V5-V6 (←), and an RSR′ pattern in lead V1 (→). It is possible that some of the precordial leads were placed in the wrong position on **ECG 117A**, accounting for the difference in the QRS complex morphologies in leads V1-V2 between the two ECGs. The QRS complex axis and duration and QT/QTc intervals are the same as in **ECG 117A**. As a result of variable AV block, clear flutter waves can be seen in leads II, III, and aVF (+). The atrial rate is 320 bpm, confirming that the rhythm in **ECG 117A** is atrial flutter. The variable block is possibly the result of electrocardioversion, which causes an increase in vagal tone as a result of the delivery of energy to the heart. The increase in AV block may also be the result of the increased sedation achieved with propofol. ■

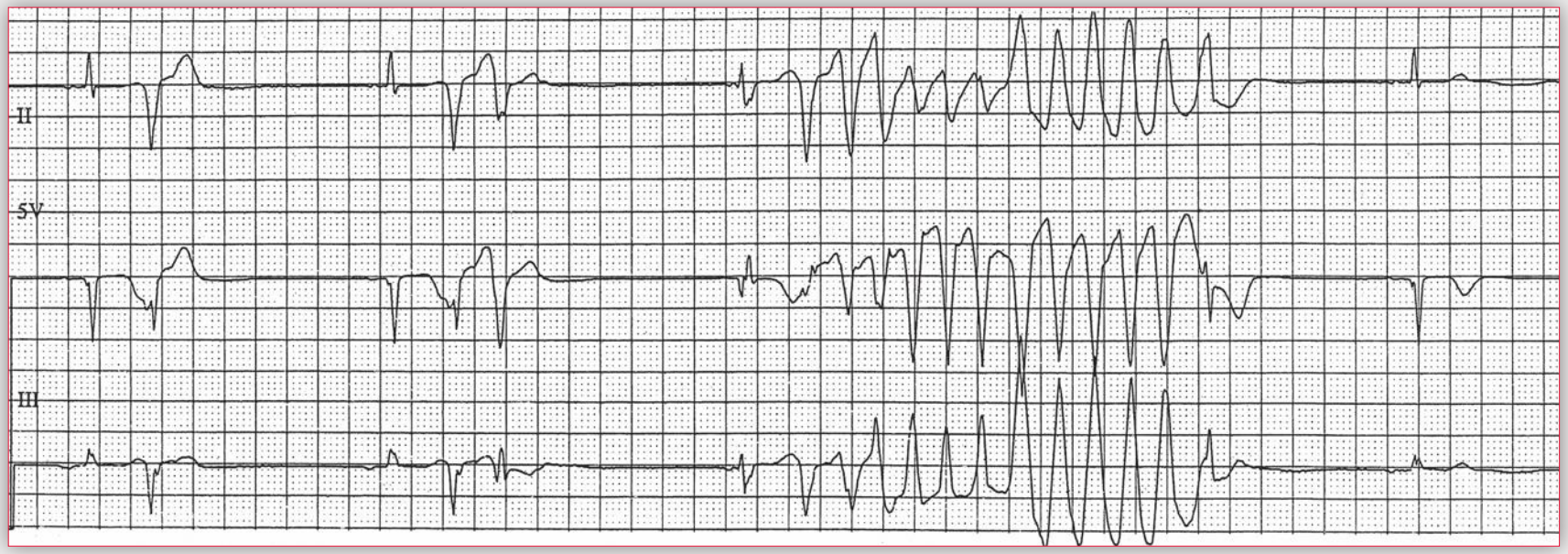
Notes

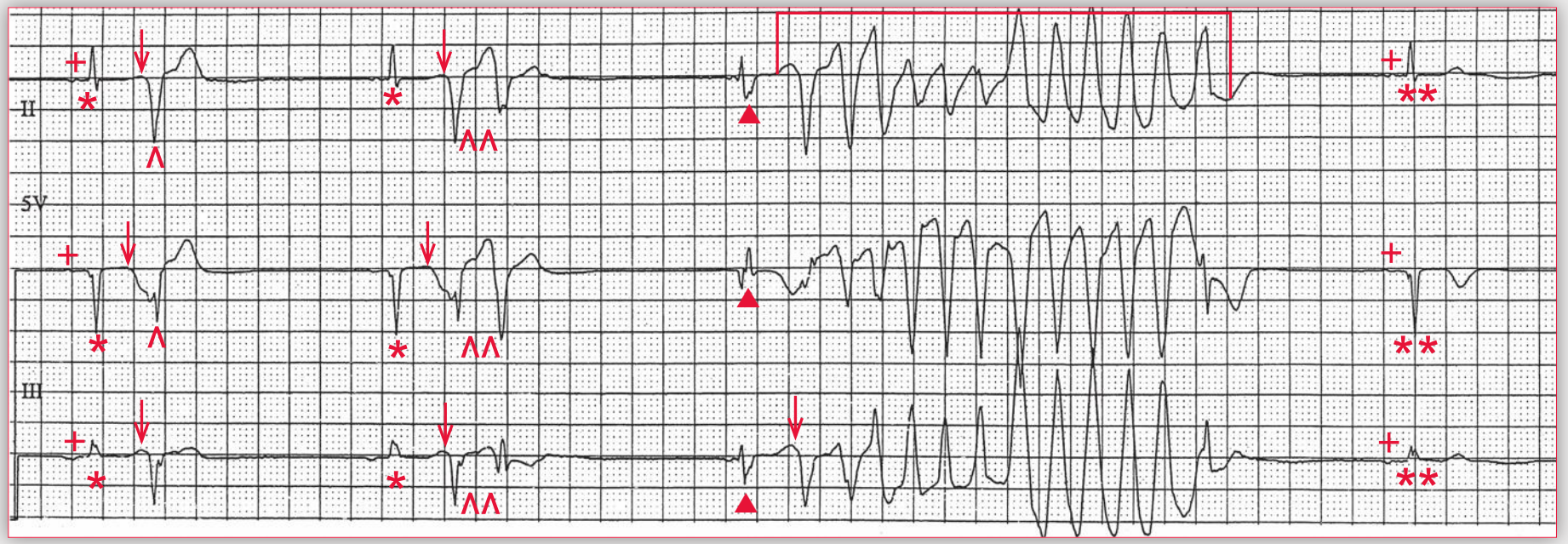
Practice Case 118

A 57-year-old woman presents to the emergency department with symptoms of chest pressure at rest, lasting for about 1 hour. She has a history of left main coronary artery stenosis as well as three-vessel disease for which she underwent bypass surgery 4 years ago. An ECG (not shown) gives no evidence of acute changes, and cardiac biomarkers are normal. She is treated with intravenous nitroglycerin and heparin, which relieve her symptoms, and admitted to the coronary care unit for observation. Three hours after admission she again complains of chest discomfort, and the nurse notices an abnormality on the bedside monitor.

What does the monitor recording show?

What is the appropriate therapy?





ECG 118 Analysis: Normal sinus rhythm, R-on-T premature ventricular complexes, nonsustained polymorphic ventricular tachycardia

This telemetry strip was recorded during an episode of chest pain. Initially two narrow QRS complexes can be seen (*), both of which are preceded by P waves (+) with a constant PR interval of 0.14 second. The last QRS complex (**) is also narrow and is preceded by a P wave with the same PR interval (0.14 sec). Importantly, the QT interval is normal (420 msec). There is a single premature ventricular complex (PVC) (^) after the first narrow QRS complex. The interval between this PVC and the preceding supraventricular complex is short; the PVC occurs slightly after the apex of the T wave, on the early downslope (↓). This is, therefore, an R-on-T PVC. After the second narrow QRS complex there are two sequential PVCs (^^), termed a ventricular couplet. As before, it is occurring on the downslope of the T wave (↓). There is a third narrow QRS complex (▲), which is again followed by an R-on-T PVC. (↓) However, this PVC provokes a brief episode of polymorphic ventricular tachycardia (⊓). This tachycardia has QRS complexes that are changing in morphology and axis. Because it is self-terminating, this is termed nonsustained polymorphic ventricular tachycardia.

Polymorphic ventricular tachycardia is seen in two situations and depends on the baseline QT interval. If the QT interval of a sinus or supraventricular complex is normal, it is polymorphic ventricular tachycardia, which is associated primarily with ischemia. Less commonly seen is polymorphic ventricular tachycardia and a normal QT interval associated with a congenital or familial condition known as catecholaminergic polymorphic ventricular tachycardia. These patients have a structurally normal heart but have a genetic mutation of either a ryanodine or calsequestrin 2 gene. ■

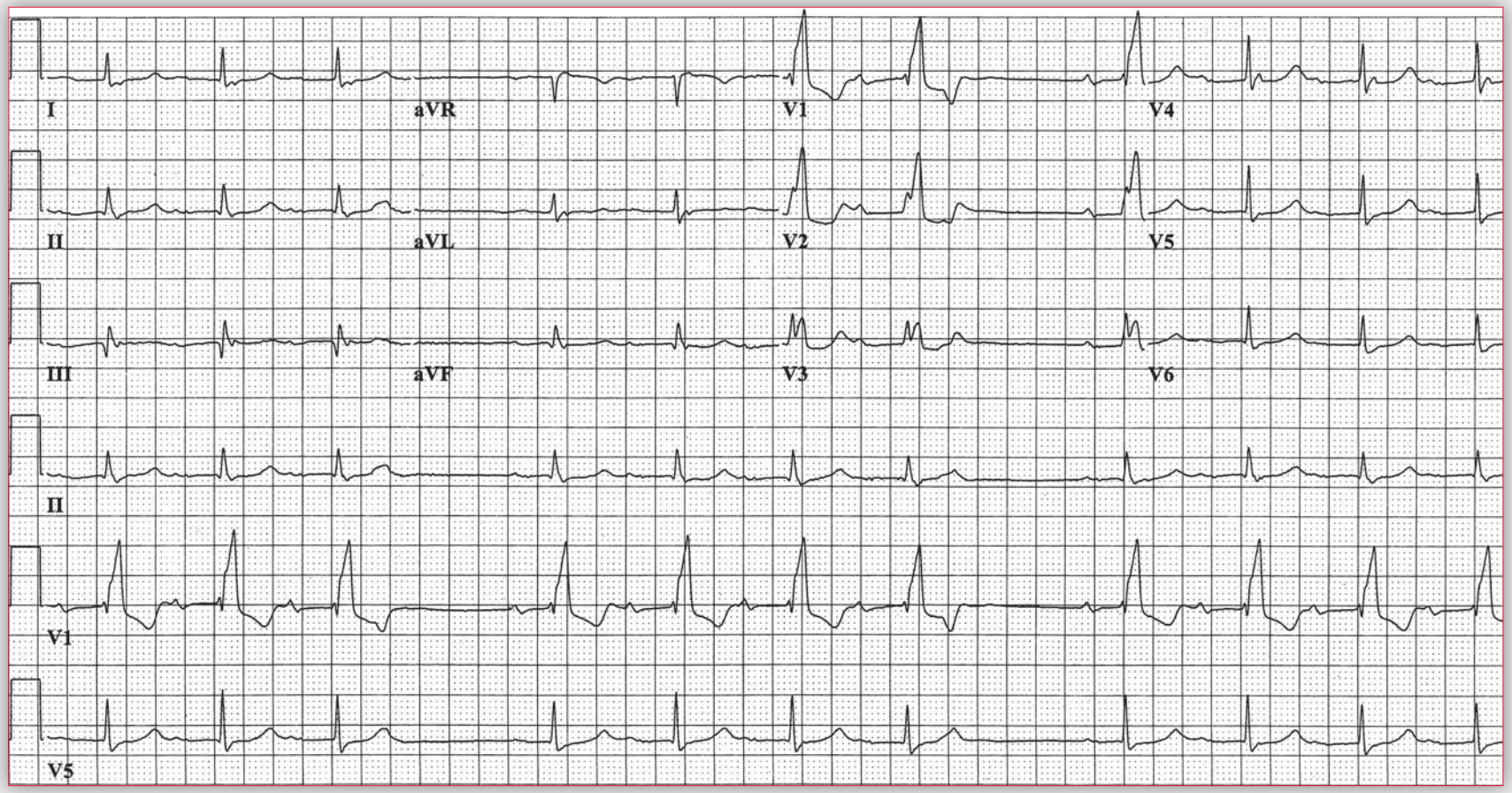
Notes

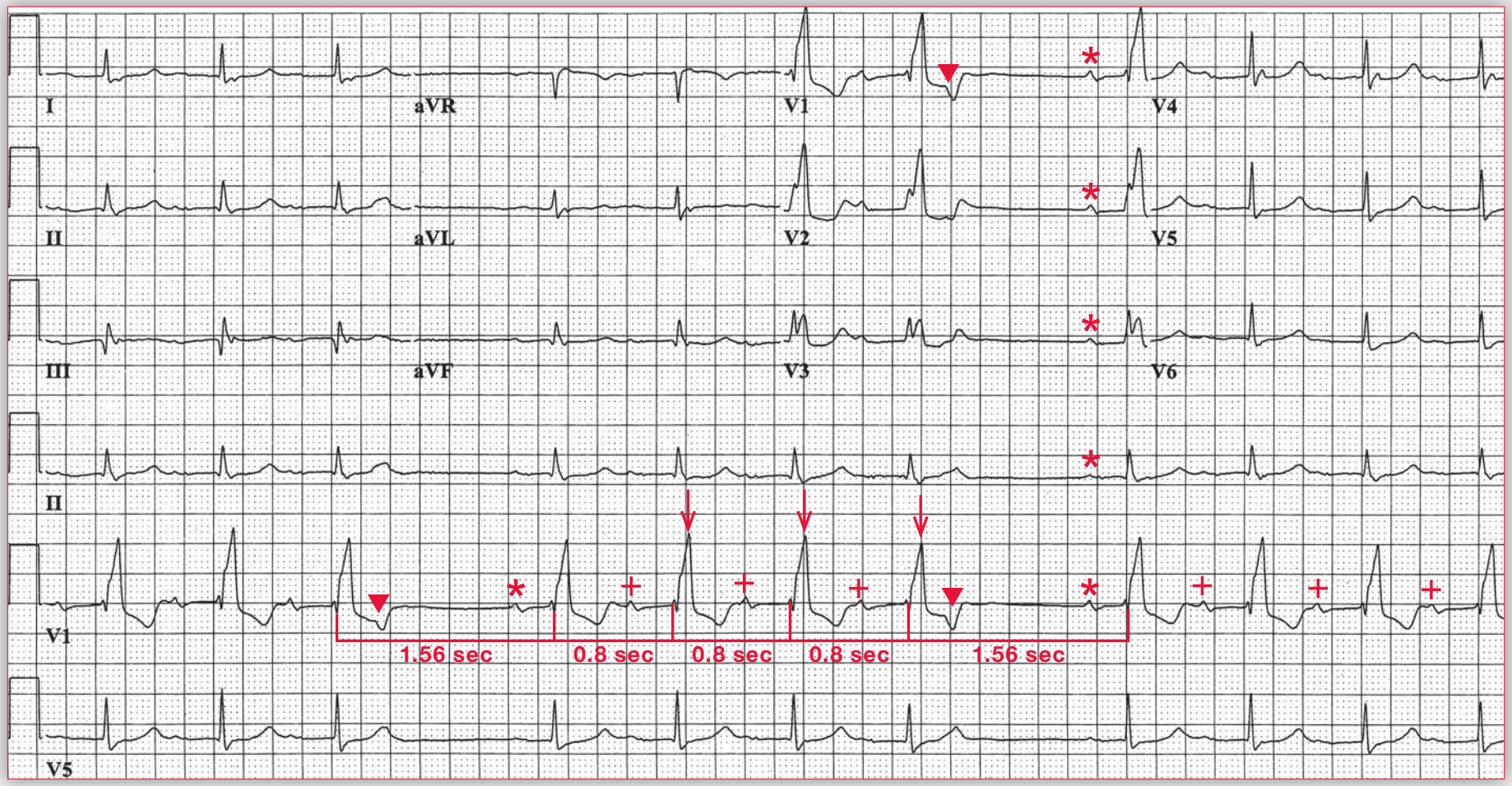
Practice Case 119

A 68-year-old man with severe sleep apnea is admitted to the surgical service following a cholecystectomy. Frequent "dropped beats" are noted on telemetric monitoring.

What does his ECG show?

What is the cause of the dropped beats?





ECG 119 Analysis: First-degree AV block, nonconducted premature atrial complexes, atrial premature beats, right bundle branch block

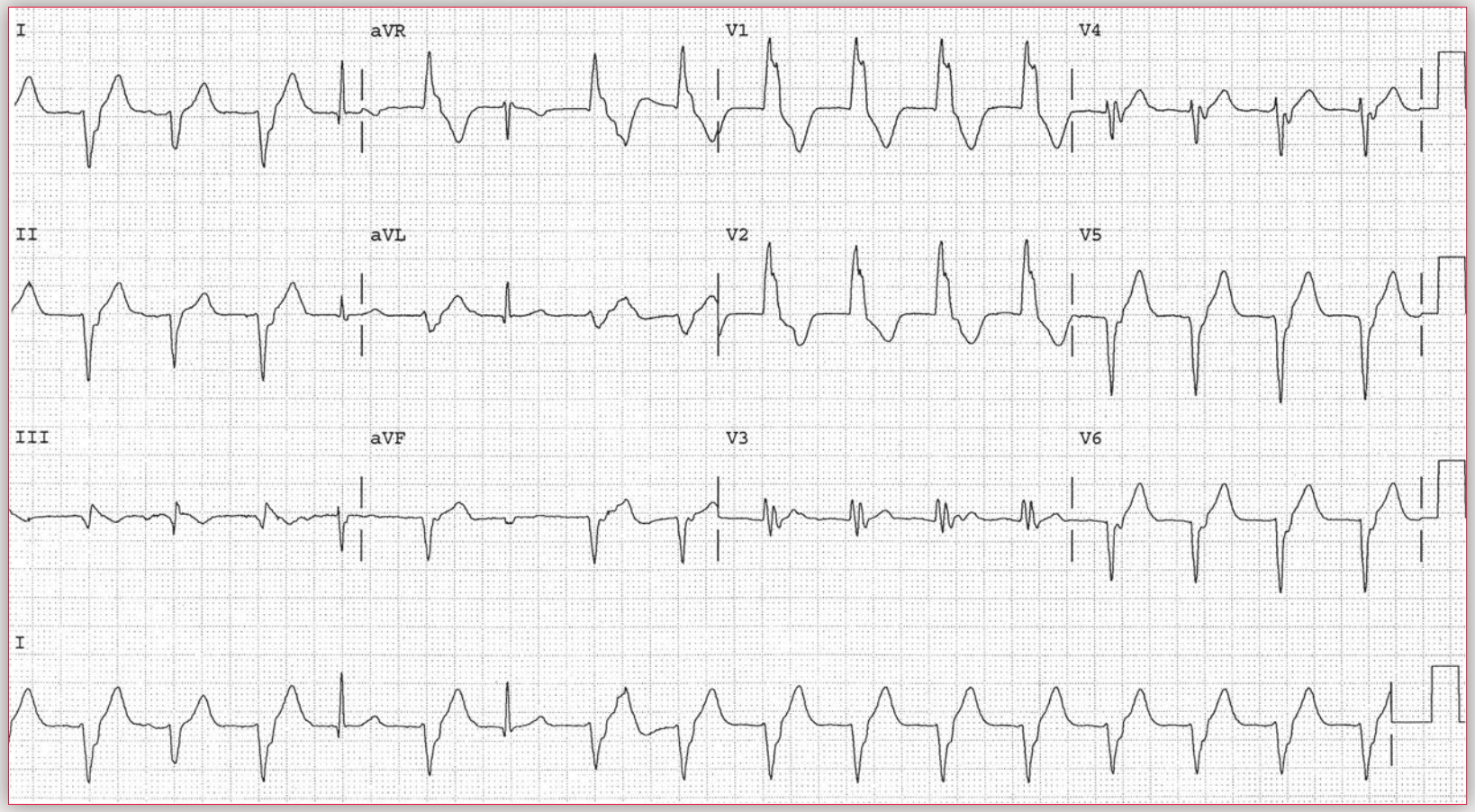
There is a regularly irregular rhythm with what looks like grouped beating. After each long RR interval there is a P wave (*) that is of the same morphology. Following this are three QRS complexes (↓), each of which is the same and is preceded by a P wave (+) with the same morphology. Thus all the P waves have the same morphology. The first PR interval after the pause is slightly shorter (0.28 sec) than the PR intervals of the subsequent QRS complexes (each 0.32 sec). The shorter PR interval may be the result of enhanced AV nodal conduction as a result of the long pause or RR interval or may represent a junctional escape complex. The P wave is positive in leads I, II, aVF, and V4-V6; hence this is a normal sinus rhythm at a rate of 74 bpm with first-degree AV block. The last QRS complex just prior to the long RR interval or pause contains a T wave that is different in lead V1 (▼); it is deeper and had a peaked appearance. Notching of the ST segment can be seen in lead V3. This is the result of a superimposed P wave that is premature (*ie*, a premature atrial complex [PAC]). This PAC is nonconducted or blocked, resulting in a pause or a long RR interval.

The QRS complex is widened (0.16 sec), and it has a pattern of a right bundle branch block (RBBB; RSR′ morphology in lead V1 and a broad S wave, which is almost isoelectric, in leads I and V5-V6). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/440 msec and 340/380 msec when corrected for the prolonged QRS complex duration). In this patient the nonconducted premature P waves may be the result of the first-degree AV block, which likely is caused by an AV nodal abnormality (*ie*, slow conduction through the node). As a result the AV node is not capable of conducting impulses at a rapid rate and hence premature P waves, which are at a rapid rate with a short PP interval, are unable to conduct through the AV node. The AV nodal abnormality as well as the RBBB may be caused by the patient's longstanding obstructive sleep apnea with possible pulmonary artery hypertension and right ventricular hypertrophy. The presence of right ventricular hypertrophy cannot be diagnosed on the ECG in the presence of a RBBB. ■

Practice Case 120

A 56-year-old diabetic man with no known heart disease presents with near-syncope. His blood pressure is 100/60 mm Hg, and he is diaphoretic. You obtain an ECG (120A). ECG 120B is his baseline ECG.

ECG 120A

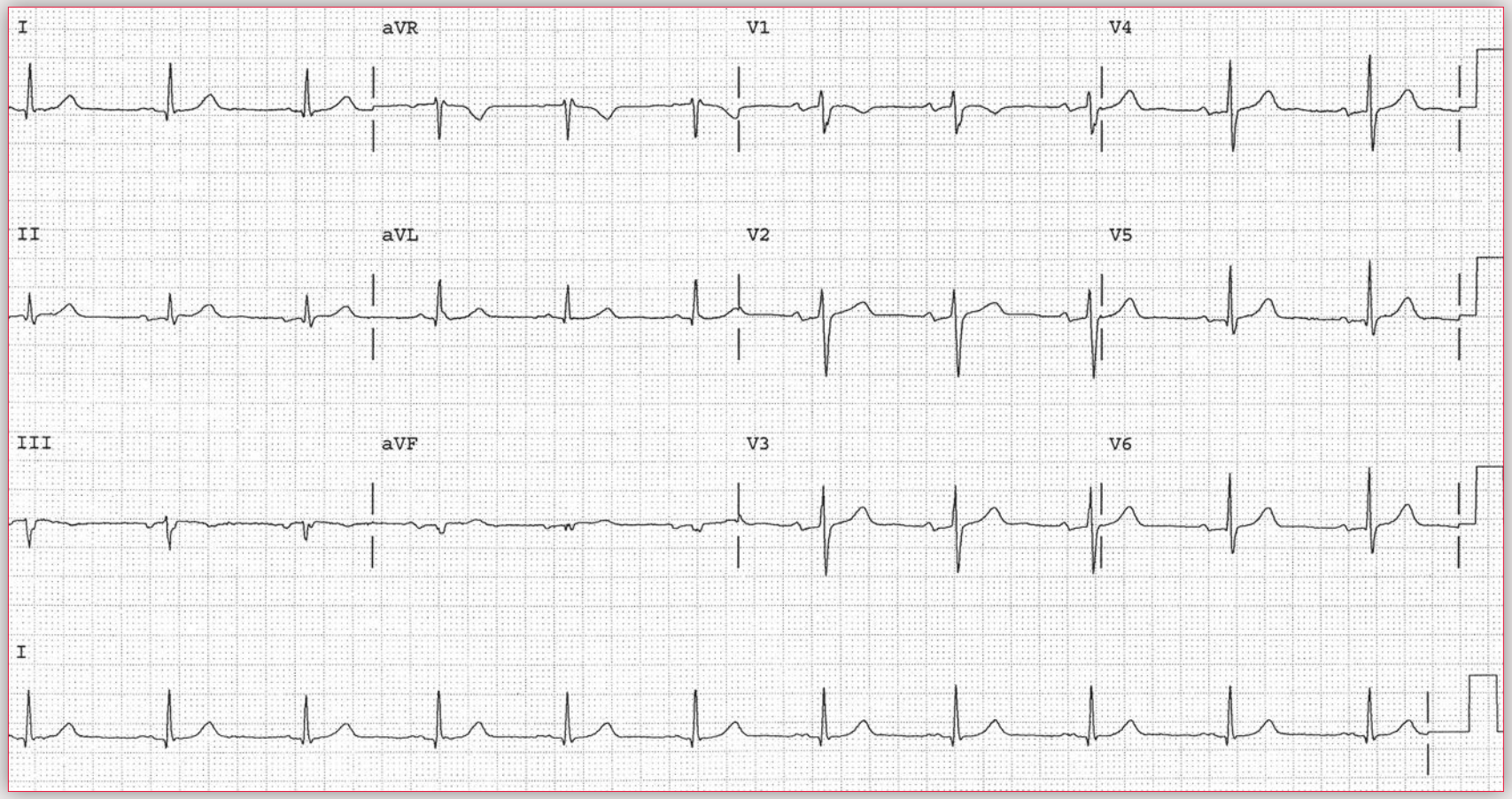


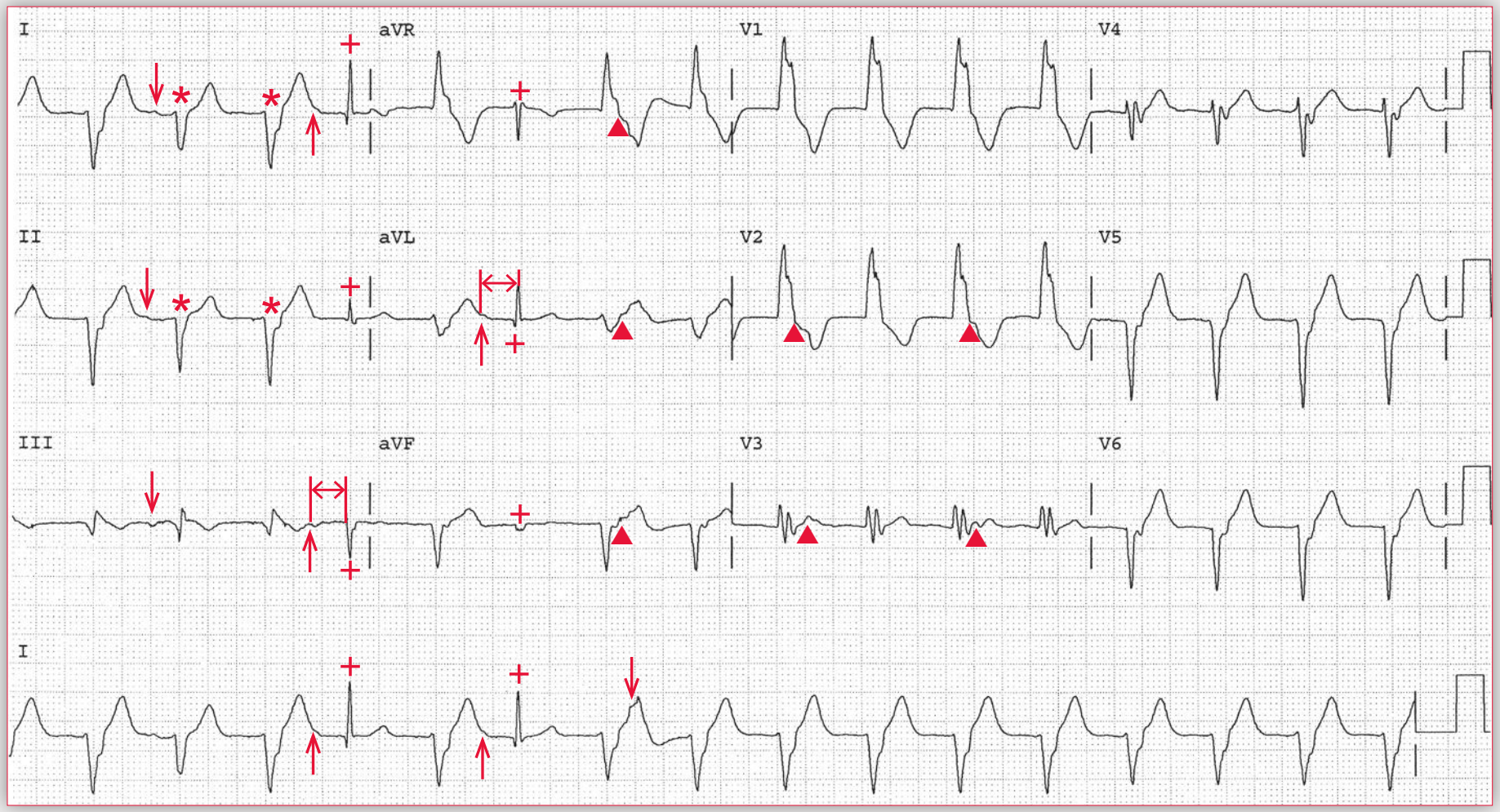
Practice Case 120

What is the rhythm abnormality?

How would you evaluate the etiology of this arrhythmia?

ECG 120B





ECG 120A Analysis: Sustained monomorphic ventricular tachycardia, Dressler (capture) complexes, AV dissociation

ECG 120A shows a regular rhythm at a rate of 100 bpm. The QRS complex duration is prolonged (0.16 sec) and the axis is indeterminate, between –90° and +/-180° (negative QRS complex in leads I and aVF). Two narrow QRS complexes (+) (duration 0.08 sec) are preceded by P waves (↑) with the same PR interval (↔) of 0.24 second. These are, therefore, sinus complexes. Although no other P waves are clearly obvious, there appears to be a P wave (↓) in leads I and III between the first and second QRS complexes. Also noted are ST-T wave irregularities, especially obvious in leads V2-V3 (▲), that are suggestive of P waves. In addition, there are subtle differences in QRS complex morphology (*), especially obvious in leads I and II. These findings indicate AV dissociation, and the narrow sinus complexes (+) represent intermittent capture or Dressler complexes. Hence this rhythm has a number of features consistent with a diagnosis of sustained ventricular tachycardia; that is, the presence of AV dissociation is confirmed by the occurrence of occasional captured sinus beats (Dressler complexes) and probably a fusion beat (second QRS complex, which is preceded by a P wave [↓] with a short PR interval and a slight change in QRS complex morphology).

Captured and fusion complexes result from an impulse conducting through the AV node that fuses with an impulse generated in the ventricular myocardium. Two other features confirm ventricular tachycardia. First is the presence of an indeterminate axis, which indicates that impulse conduction is not via the normal His-Purkinje system but results from direct myocardial activation. This is seen with a ventricular complex, paced complex, or preexcited complex (*ie,* Wolff-Parkinson-White). **Second**, non–rate-related changes in ST-T waves and slight changes in QRS complex morphology can confirm ventricular tachycardia. The subtle changes in QRS complex morphology and ST-T waves are due to the fact that with ventricular tachycardia ventricular activation is not via the normal His-Purkinje system (in which case the fixed pathway for activation would result in uniform QRS complexes) but is by direct myocardial activation (in which the pattern of conduction may change). The changes in the ST-T waves reflect changes in ventricular repolarization or superimposed P waves. Importantly, any superventricular rhythm (sinus, atrial, or AV nodal) always conducts to the ventricle through the same pathway; hence the QRS complexes and ST-T waves are identical. As all of the QRS complexes are similar in morphology (although with slight changes in appearance), this is monomorphic ventricular tachycardia.

This wide complex tachycardia has a QRS complex morphology that resembles a right bundle branch block (R-wave amplitude taller than S-wave depth in lead V1). The lack of an RSR′ pattern in lead V1 and the presence of S > R in lead V6 are both consistent with the diagnosis of ventricular tachycardia.

Monomorphic ventricular tachycardia is associated with chronic ischemic heart disease (although not active myocardial ischemia but rather due to scar or fibrosis), cardiomyopathy of any etiology (*eg*, hypertrophic, infiltrative, or dilated cardiomyopathy), congenital syndromes (*eg*, Brugada or arrhythmogenic right ventricular cardiomyopathy), outflow tract tachycardias typically in the setting of a structurally normal heart, electrolyte imbalances, and drug toxicity

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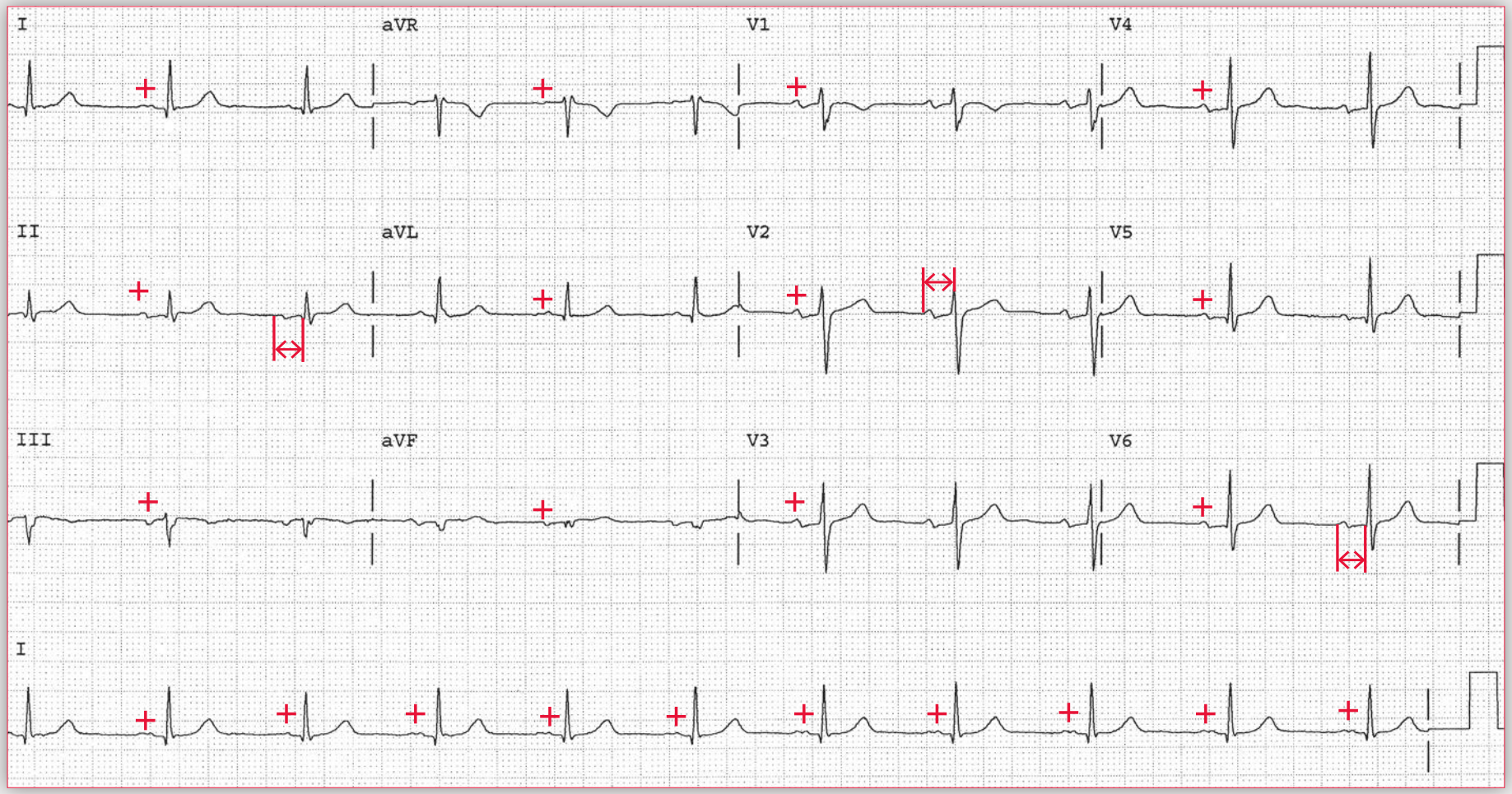

ECG 120B Analysis: Normal sinus rhythm

(*ie*, cocaine, digitalis). Hence, besides routine blood work and toxicology screening, the evaluation for ventricular tachycardia should include an evaluation for coronary artery disease with some form of stress testing (treadmill, pharmacologic, or exercise echocardiography). Echocardiography can be used to assess for structural heart disease that may be linked to ventricular tachycardia, such as valvular disease, chamber enlargement, or myocardial hypertrophy. Holter monitoring can be used to assess the frequency and duration of ventricular tachycardia episodes. Magnetic resonance imaging is often used to assess for areas of ventricular fibrosis, inflammation, or fatty infiltration.

ECG 120B shows a regular rhythm at a rate of 64 bpm. There is a P wave (**+**) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I and V4-V6 and biphasic (positive–negative) in leads II and aVF. Hence this is a sinus rhythm. The QRS complex duration is normal (0.08 sec), and it has a normal morphology. The QT/QTc intervals are normal (400/410 msec). The QRS complex duration and morphology are identical to the narrow and conducted QRS complexes seen in **ECG 120A**. This confirms the fact that the narrow QRS complex in ECG **120A** are indeed intermittent captured or Dressler complexes.

However, the PR interval (↔) is shorter than that seen with the conducted complexes (Dressler complexes) in **ECG 120A**. This is the result of retrograde concealed conduction. During ventricular tachycardia, there is retrograde (ventriculoatrial) impulse conduction into the AV node from the ventricular complex. However, if the retrograde impulse conduction results in only partial penetration of the AV node and the impulse is extinguished within the node (*ie*, the retrograde conduction is concealed). the AV node is only partially depolarized and the next sinus impulse may get through in an antegrade direction (hence AV conduction) but at a slower rate. This will result in a longer PR interval. The difference in the PR intervals between the Dressler complex (0.24 sec) and the sinus complex (0.20 sec) is, therefore, a result of retrograde concealed conduction into the AV node from the ventricular tachycardia.

Most often a faster ventricular rhythm results in repeated and complete retrograde (ventriculoatrial) activation of the AV node, rendering it continuously refractory to sinus impulses and antegrade (AV) conduction. This accounts for the AV dissociation frequently seen with ventricular tachycardia as the node is generally refractory and does not conduct the sinus impulse in an antegrade fashion. However, if an appropriately timed sinus impulse enters the AV node antegradely before the ventricular impulse completely penetrates and depolarizes the AV node retrogradely (*ie*, is concealed within it), the AV node is not totally refractory and may conduct the sinus impulse, although at a slower rate as it is partially refractory. This results in a longer PR interval when compared with the PR interval of the sinus complex. ■